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The University of Toronto Medical Journal

(AN UNDERGRADUATE PUBLICATION)

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Editorial

Salvete '46

It is our privilege to welcome the class of '46 into the Faculty of Medicine, on behalf of the students and Medical Society.

We can recall very vividly when we, too, entered Medicine, when University was a new and strange experience, and when Graduation seemed very distant. But this we say to you, though it sound like a platitude, the years will pass quickly, and in all too short a time your years for learning will have ended, and you will go out into the world "on your own".

Anyone who enters Medicine takes upon his shoulders a responsibility. For those who enter in 1941 it is even greater. You come at a time when every man and woman is expected to put forth the best effort to further the cause. You have chosen to do so through the medium of medicine. See to it, then, that your time be not wasted. Study hard, play hard, but don't overdo either, since health, both physical and mental, is essential for the tasks in hand.

It has come to the knowledge of the Medical Society, that medical students are being accosted in the streets and other public places, and being insulted for not being in uniform. This is a trend which we do not like to see. Doubtless other people, such as those working in essential industries, are being subjected to the same

treatment. We wish to assure the students that the Medical Society is aware of the situation and has taken such steps as are in its power to try to put the matter right.

Many will notice, in fact, few can help noticing, that the JOURNAL has blossomed forth in colour. This follows the adoption of a policy in which the colour of the cover will be changed each year. It is hoped that this will help students to keep the yearly sets separate, and facilitate the finding of articles. The size of the JOURNAL and the cover design will remain unchanged.

In Memoriam

GEORGE M. BIGGS, M.B.

Professor and Head of the Department of Oto-Laryngology

Those who knew well, and over many years, the late head of the Department of Oto-Laryngology, feel that the University, the Toronto General Hospital, his friends and the community, lost, at the beginning of this medical term, one who gave evidence of an unusual combination of qualities of value. He early showed prowess in athletics and was for several years on Varsity First Rugby and Varsity Hockey teams.

After Post Graduate study in his specialty at the University of Vienna in 1913 and 1914, he soon became known in Toronto for his balanced judgment and decisions, never hurried except in emergency. Under any circumstances he always "knew what time of day it was", he never lost his perspective. In play or work, alongside of a quiet and modest persistence, and whenever occasion by a lull might permit, there would appear an unobtrusive humour, if only by a twinkle or a grunt. Those who knew George Biggs loved him.

W. E. O.

PILONIDAL CYSTS AND SINUSES: ETIOLOGY AND TREATMENT

By DR. JAMES T. DANIS, F.R.C.S.(Edin.), F.A.C.S.
Demonstrator in Surgery, University of Toronto

Synonyms Postanal, sacrococcygeal, coccygeal dimple, sinus or fistulae
Sequestration, coccygeal, postsacral, traction dermoid.
Foveola coccygea.

Few subjects in Surgery have given rise to more controversy as to etiology and more disappointments as to treatment than the one now to be considered. I shall attempt in this article to crystallize these views and arrive at definite conclusions.

Etiology

Three main theories have been suggested to explain this most interesting condition.

1. *Figure 1.* That it is due to some derangement in the development of the neural tube at its caudal end. It will be recalled that the neural plate which serves to form the neural groove and later the neural or medullary tube, actually the future spinal cord, is derived from ectoderm. During the early weeks of embryonic life, the medullary tube extends to the tip of the coccyx. At this time the muscle elements of the mesoderm approach the midline at the back except in the sacrococcygeal region where they are crowded to the side, resulting in closer approximation of the neural tube to the skin, which has now fused in the midline over the submerged tube. As the vertebral column grows more rapidly than the medullary tube, considerable inequality exists, the latter and the overlying skin are placed on the stretch. This stretched portion of the tube becomes divided into two segments, a direct coccygeal and a reflected caudal segment. The coccygeal segment becomes absorbed while the reflected caudal portion adherent to the skin may persist normally as a dimple or give rise

to mixed tumors. The adherents of this theory suggest that pilonidal cysts and sinuses arise from a derangement in the development of this caudal segment.

2. That it is due to the persistence of some portion of the neurenteric canal, a communication which in the early embryos normally exists between the neural groove and hind-gut. See *Figure 1*.
3. Followers of the third theory, and the one now generally accepted and to which I humbly pledge my allegiance, believe that it has nothing to do with a failure of the development of the neural tube or neurenteric canal, but owes its presence to a faulty coalescence of the ectoderm in the midline over the sacrococcygeal region. The condition is characterized by the formation of one or more tracts or cysts in which is collected the product of dermal activity, sometimes including hair, with a tendency to burrow and undergo abscess formation. These abscesses rupture through the original or primary site of invagination which is always in the midline, shown in *Figure 2*, or through secondary openings to either side of the midline, *Figure 3*. This plate shows four primary openings in the midline indicated by probes and one secondary opening to the right of the midline. This dotted line indicates the probable subcutaneous extent of the disease. Note the small swelling adjacent to secondary sinus representing an abscess.

Symptomatology

Although the disease is developmental in origin, a pilonidal sinus or cyst causes

no symptoms unless infection has occurred, in which event the complaints are usually those of recurrent abscess occurring spontaneously or following an exciting cause such as a fall or blow. If these abscesses have ruptured spontaneously, a tuft of granulation tissue will indicate its former site, but if they have been incised, linear scars are visible.

Treatment

The successful treatment of a pilonidal sinus is surgical, and consists of excision of all cysts and sinuses and

their ramifications. If an abscess develops this must be incised and allowed to shrink before any attempt is made to excise the tracts. Indeed, an abscess may be the first indication of the trouble. Before operation for radical cure, these cases undergo a routine proctoscopic examination to rule out the possibility of some associated rectal pathology. I do not advocate too vigorous probing as it is painful and there is danger of carrying the infection beyond the limits of the cyst walls. The tracts are injected with lipiodal under gentle pressure and antero-posterior and lateral plates are

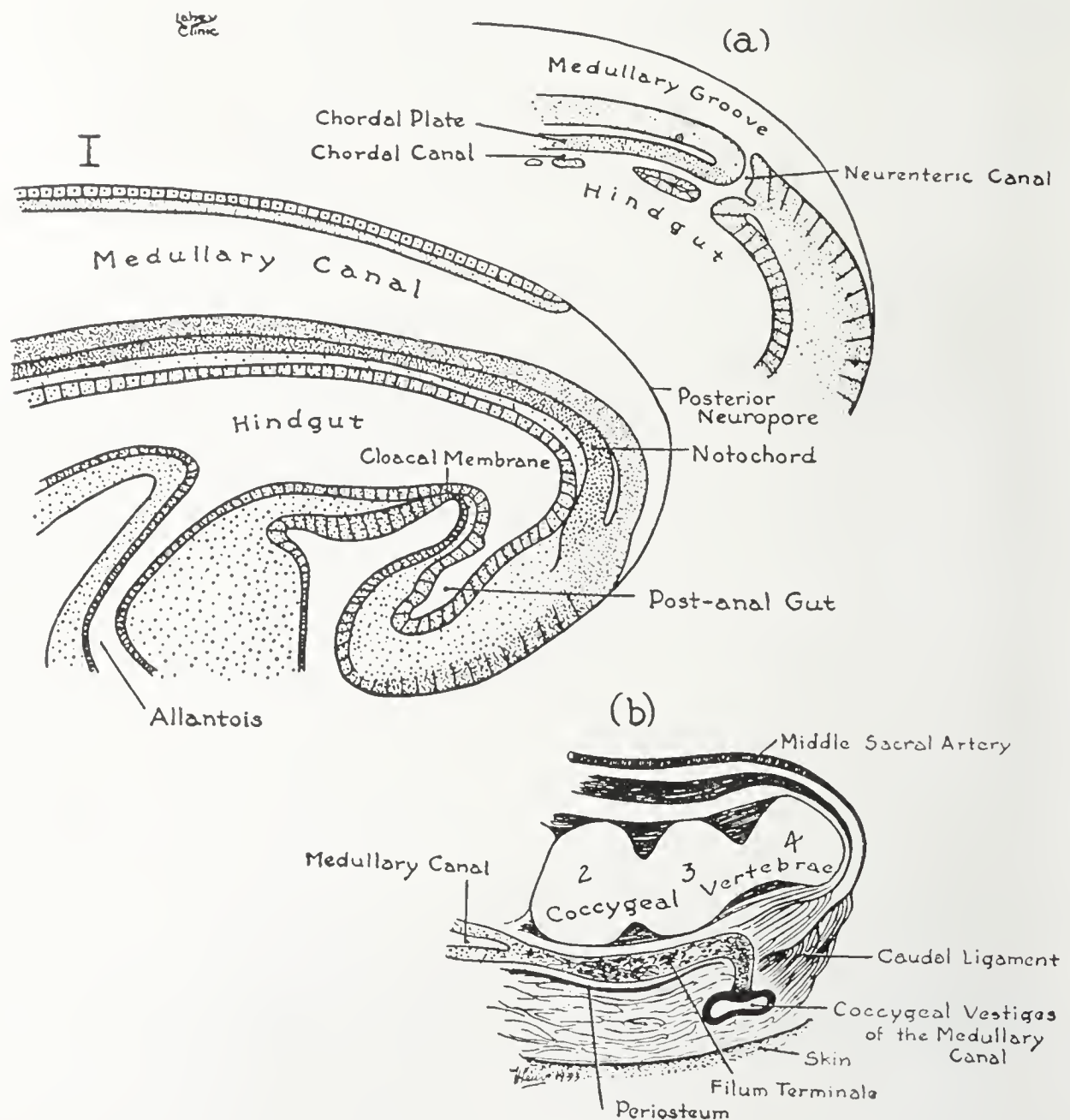


FIG. 1.—I. Median section through caudal end of Eternod's embryo with eight pairs of somites. a. Median section through caudal end of Bremer's 4mm. embryo. b. Schematic median section through caudal end of a human fetus 11 cm. long. (After Unger and Brugsch.) Note that anus will be formed by absorption of cloacal membrane anterior to distal end of gut, leaving "post-anal gut" to become obliterated. In earlier embryo (a) is shown position of the neurenteric canal. Note in (b) vestiges of distal end of medullary canal left near tip of coccyx, and "caudal ligament." (After Keibel and Mall.) (Courtesy Lahey, F. H. and Eckerson, E. B., The American Journal of Surgery, January 1934, Volume XXIII, Page 31.)



FIG. 2.—Photograph showing a pilonidal sinus secondarily infected, with a granuloma presenting in one of the openings of the sinus. In this case the abscess was located in the superficial cystic dilatation of the sinus.

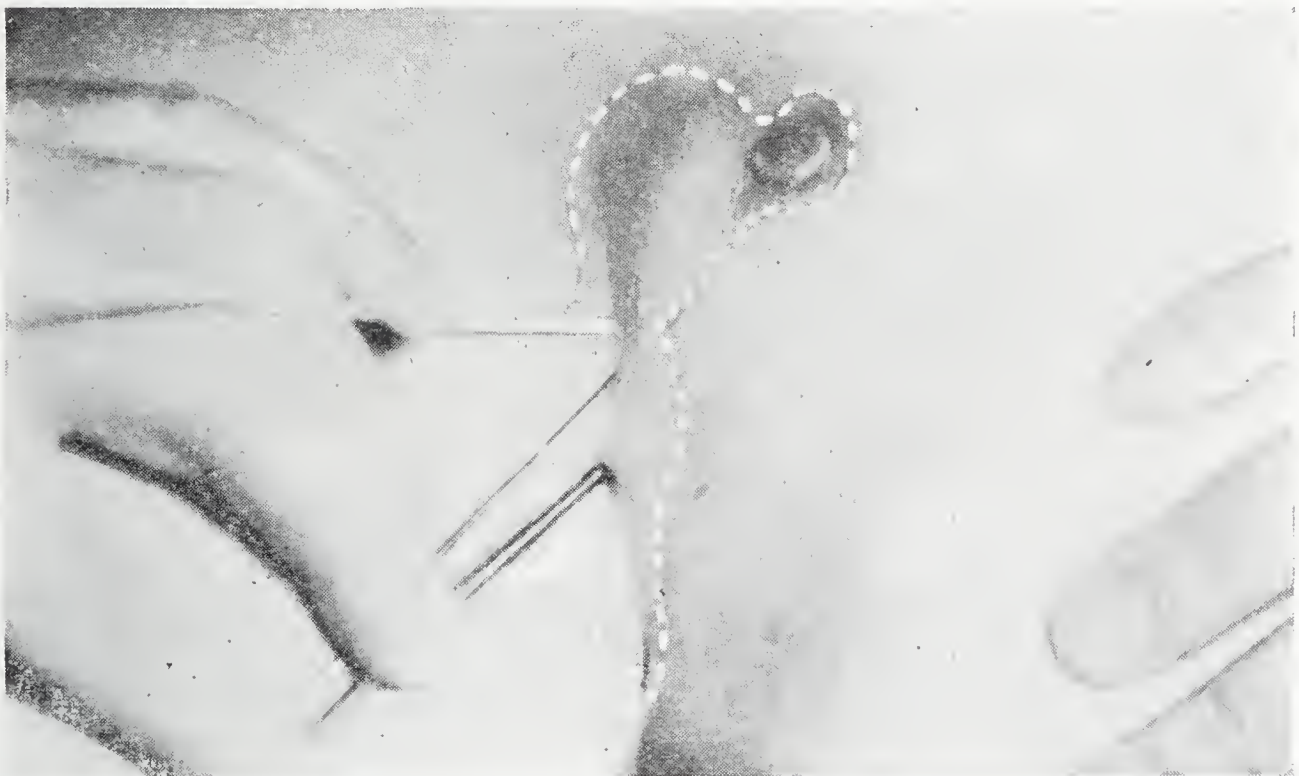


FIG. 3.—Photograph showing a deep seated cystic dilatation of a pilonidal sinus with secondary sinus formation. There are four primary openings and one secondary sinus opening, the latter containing a granuloma.

made of the pelvis to show the depth and lateral limits of the cavity to be excised. It also gives us information as to the condition of the adjoining bony parts. *Figure 4* A plate in the antero-posterior direction after injection of lipiodal. It might be mentioned here that a line drawn horizontally backwards from the superior border of the symphysis pubis will pass just below the

its meninges. *Figure 6* is an antero-posterior view of another case. The opaque material extends well below and to one side of the inferior border of the symphysis. *Figure 7* is a lateral plate of the same case.

There are certain definite rules which we follow in the management of these cases. A radical excision should never be attempted in the presence of an

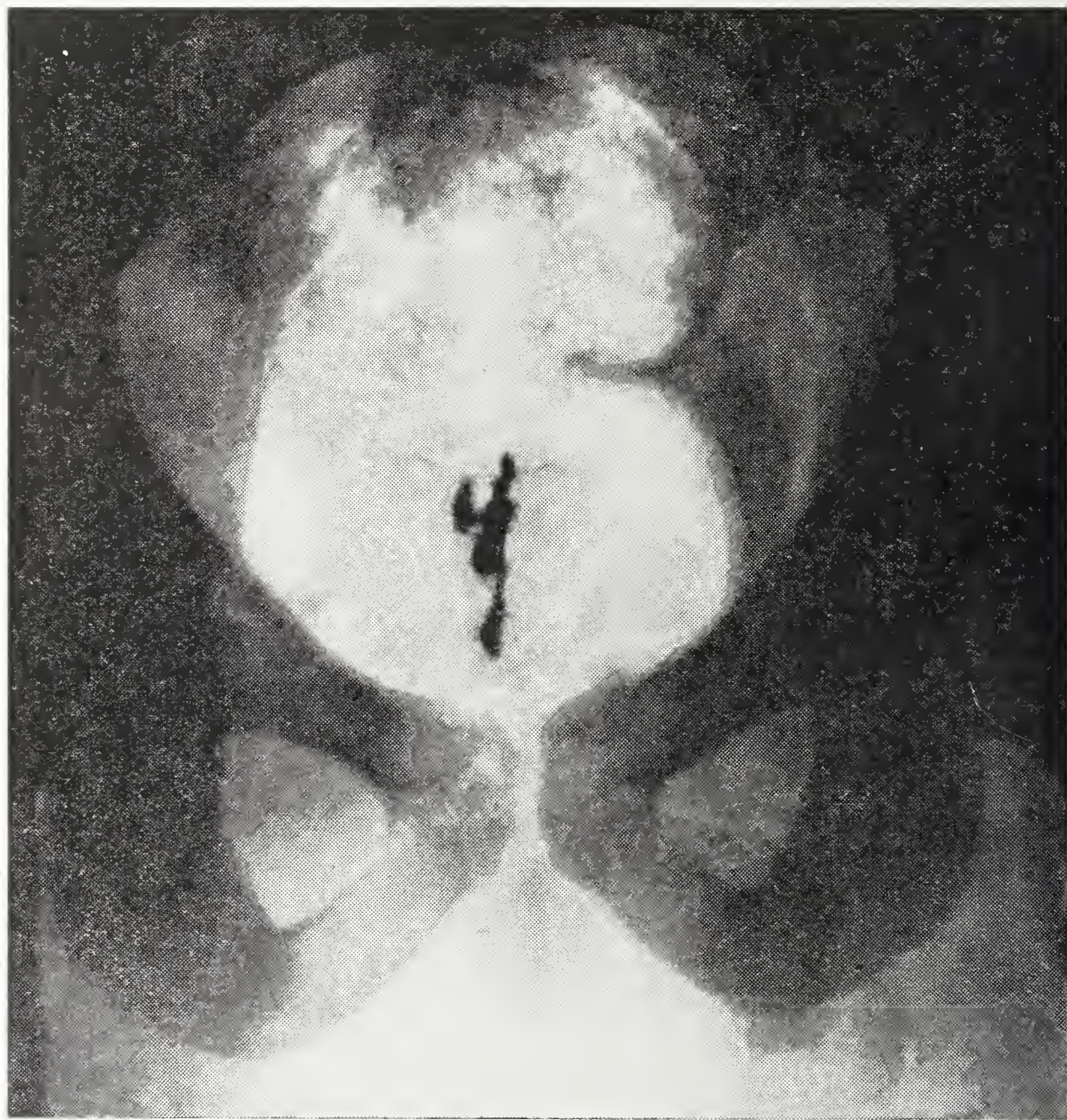


FIG. 4.—Antero-posterior plate after injection of Lipiodol.

sacral promontory, while a similar line from its inferior border will pass about half an inch above the anal orifice. It is evident that in this plate the lower limit of lipiodal is just above the sacral promontory. The importance of these landmarks will be seen when we come to consider operative treatment. *Figure 5* is a lateral view of the same case. There is no evidence that the opaque material communicates with the spinal cord or

abscess or acute infection. If an abscess is present, it is incised and allowed to shrink before excision is carried out. If the sinuses are acutely inflamed and discharging copiously, they are treated with sitz baths and warm compresses until the discharge is reduced to a minimum. Methylene blue, 10 per cent, is carefully injected into the sinus openings four or five days before operation and repeated for two or three days, giving

the patient a rest on the day prior to operation. In this manner the course of the tracts is indicated and may be of assistance at operation. Having determined the extent of the cysts or sinuses clinically and by injection of opaque material one should be able to decide whether the open or closed method will be used, although in some cases this final decision will be made at

represent the position of the primary sinuses; the black dot a secondary sinus; the oval-shaped, dotted line, the probable extent of the disease; the unbroken line, the excision. An elliptical incision is made to include all sinuses and scars from previous abscesses, plus a sufficient amount of normal tissue to insure complete removal. The base of the wound should be cleansed



FIG. 5.—Lateral plate, same patient as Fig. 4.

operation. The rule I have made in my own cases is that if the lower pole of the wound resulting from excision is within one and a half inches from the anal orifice, the closed method is not used; the danger of infection is obvious.

First I want to discuss the closed method. The patient is given a spinal anaesthetic and placed on his face in a "Jack-Knife" position. *Figure 8* The three small circles in the midline

thoroughly down to the fascia covering the sacrum and coccyx. Wound edges should be carefully examined and if there is any indication that the disease extends beyond the wound margins, shown by methylene blue or granulation tissue, more tissue is excised. The margins of the wound are next undercut over the glutei muscles. If the cavity is small it is claimed by some that it may be possible by undercutting the wound

margins, to appose the wound edges and obtain primary healing. This method of treatment, by undercutting only, is possible in a very limited number of cases where only one sinus is present, and where there is no lateral expansion of the disease. There is usually too much tension on the suture line. Disappointments with any closed method

Where primary closure is feasible, in order to avoid too much tension on the suture line and insure obliteration of dead spaces, I now use a single or double shift graft, first introduced by Lahey and later modified by Cattell of the Lahey clinic. The flap or flaps are outlined by angular incisions, beginning at least two and a half inches lateral to the

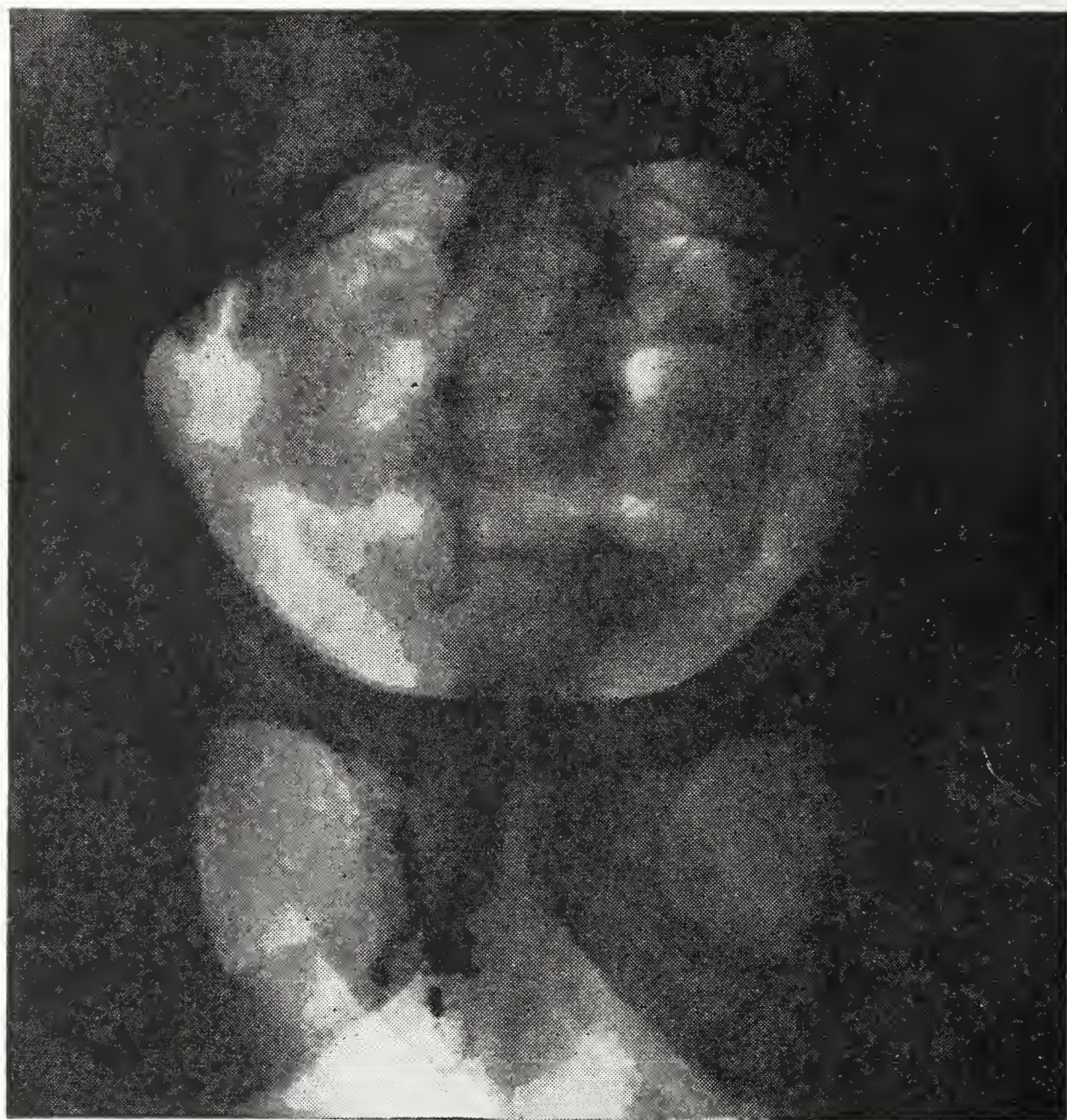


FIG. 6

of treatment may be attributed to one of the following causes, namely:

1. Excision in the presence of acute inflammation.
2. Incomplete excision of diseased tissue.
3. Faulty obliteration of dead spaces.
4. Imperfect haemostasis.
5. Tension on the suture line.
6. Sepsis following operation.
7. Use of infiltration analgesia which tends to spread infection.

upper limit of the wound in midline to insure a good blood supply, the incision going down through the full thickness of skin and fat. The lower end of this incision does not need to come down quite to the level of the wound in midline. The flaps are now undercut and sutured in the midline by figure of eight and short, silkworm gut sutures. All bleeding points are suture-ligated. Each figure of eight suture should include, if possible, the fascia over the sacrum. In

this manner the margins are apposed perfectly without tension. The angular incision is now converted into a "Y" by the introduction of short, silkworm gut sutures, figure of eight sutures not being necessary here. Two Y-shaped penrose drains are introduced under the flaps and the ends brought out under the outer corners. This is done to prevent a pool of blood from collecting under

changing dressings during the night. The dressings over the drains are changed t.i.d., those over the midline wound once daily. All dressings are changed after movements. The perianal skin is kept dry and clean. The penrose drains are shortened half an inch daily beginning the day after operation and withdrawn in two or three days. In the meantime, firm

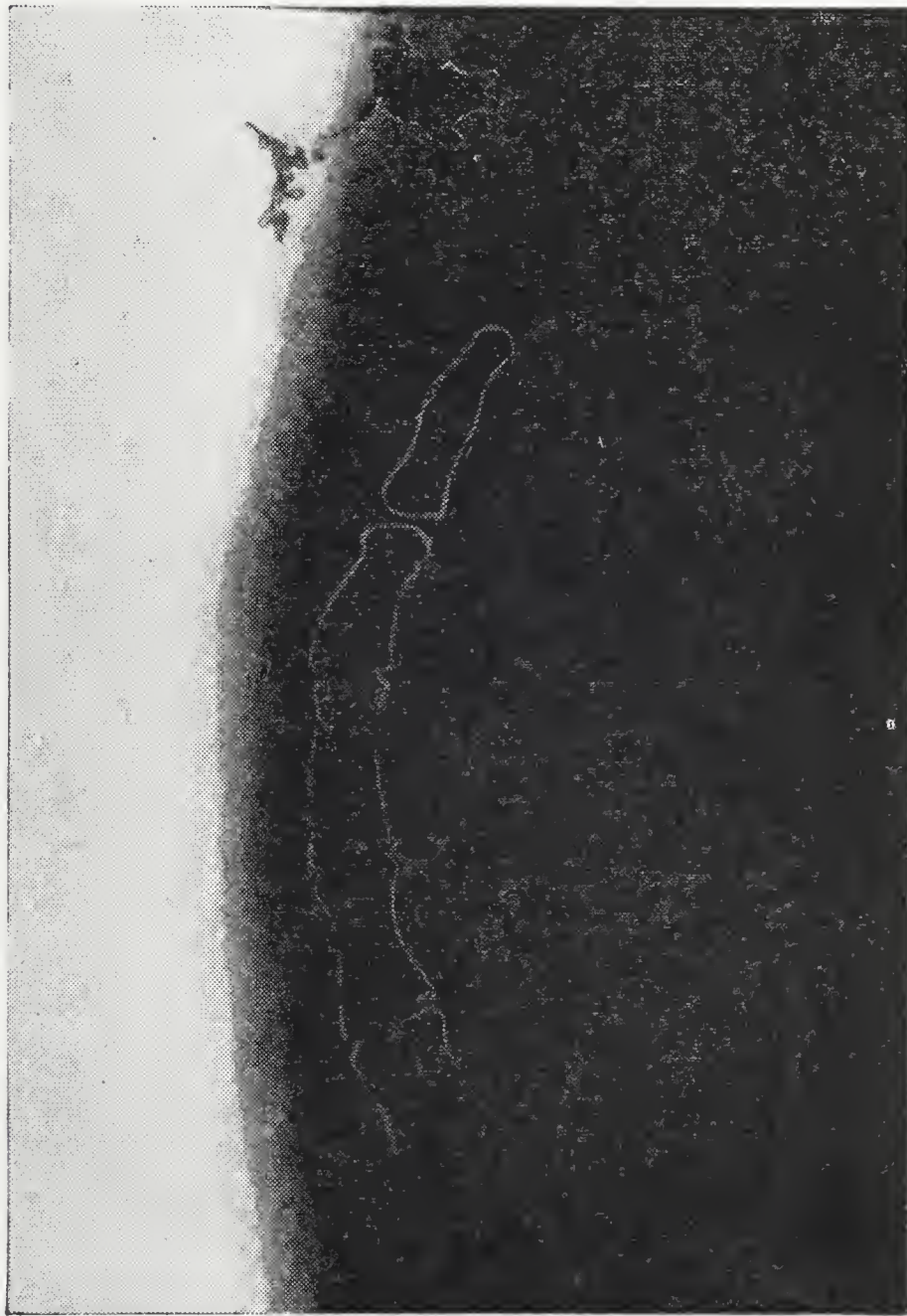


FIG. 7.—Lateral plate. Same subject as Fig. 6.

flaps. *Figure 9* represents stages of single flap repair; *Figure 10*, double flap repair.

After operation firm dressings are applied by means of diagonally applied adhesive strips and a binder is applied over this. The patient lies on his stomach or side. (It is too painful to lie otherwise.) Low residual diet for five days or more, then enemata rather than cathartics as this might necessitate

pressure must be applied continually over the dressings to ensure obliteration of the dead spaces and proper adhesion of the grafts. After the tenth day, the patient lies on his back. He may leave the hospital on the twelfth or fourteenth day after operation. The case represented in *Figures 4 and 5* was treated by the above method.

Now for the open method of treatment. I reserve the open method for

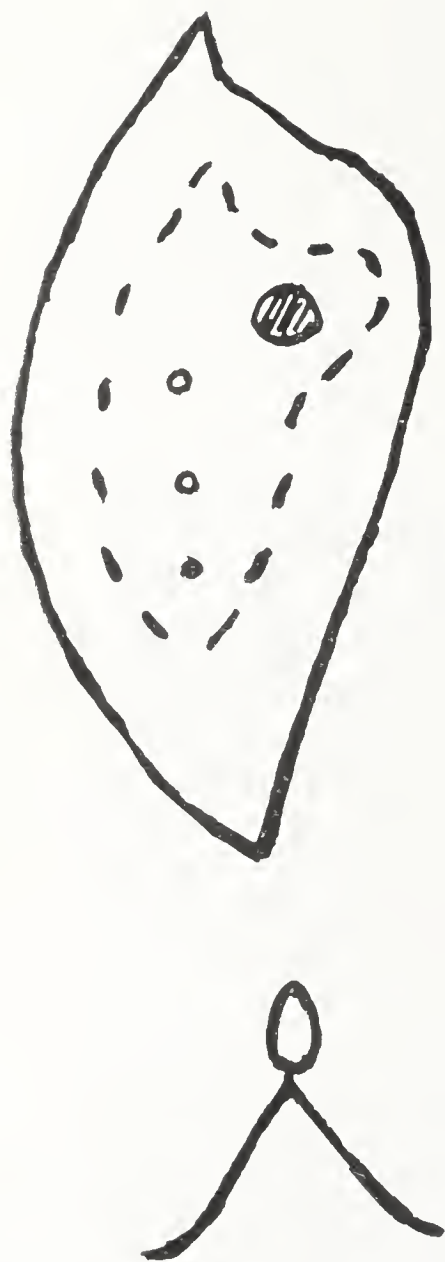


FIG. 8

cases where, on account of the close proximity of the wound to the anal orifice, the closed method is not advisable. In these open cases, especially if the intergluteal sulcus is deep, it is sometimes difficult to keep the wound edges sufficiently separated to prevent a chronic, cavernous ulcer. It is a fact, and sometimes a painful one to the surgeon and the patient's relatives, that granulating surfaces will not heal if they are approximated. We have discovered a simple method of preventing this. When granulation tissue has replaced the slough about the tenth day, the margins of the wound are drawn apart by strips of adhesive applied over a couple of special pads at the sides, and fastened over the skin in front. I have found this simple precaution very useful. The case represented in *Figures 6 and 7* was treated as above.

Synopsis

- 1. Pilonidal cysts or sinuses are due to developmental errors and owe their presence to a faulty coalescence of the ectoderm in the mid-line over the sacrococcygeal region.
- 2. They have nothing whatever to do with maldevelopment or vestigial



FIG. 9

- A—The infected area is excised in an elliptic manner and a triangular incision made to one side.
- B—By suturing the underlying tissues of the midline wound, the inner skin edge of the lateral incision is drawn medialward.
- C—Final appearance of the central and lateral wounds following complete closure.

remains of the neural tube or its meninges; neither do they originate from vestigial remnants of the neurenteric canal, notochord, post-anal gut, proctodeum or proctodeal membrane. (See again *Figure 1*).

3. Although developmental in origin they may not give rise to trouble until early adult life when they become infected or an abscess develops.
4. They are not known to become malignant.
5. From its original site in the midline the condition may spread through

repeated infection or abscess formation some distance from its initial site, leading to the development of multiple sinuses involving, in extreme cases, the ischio-rectal fossae and thereby simulating external openings of fistulae in and with which, of course, they have no connection.

6. The successful treatment is surgical and consists of complete excision of diseased tissue and all its ramifications, the resulting wound being closed by primary suture or left open depending upon its proximity to the anal orifice.

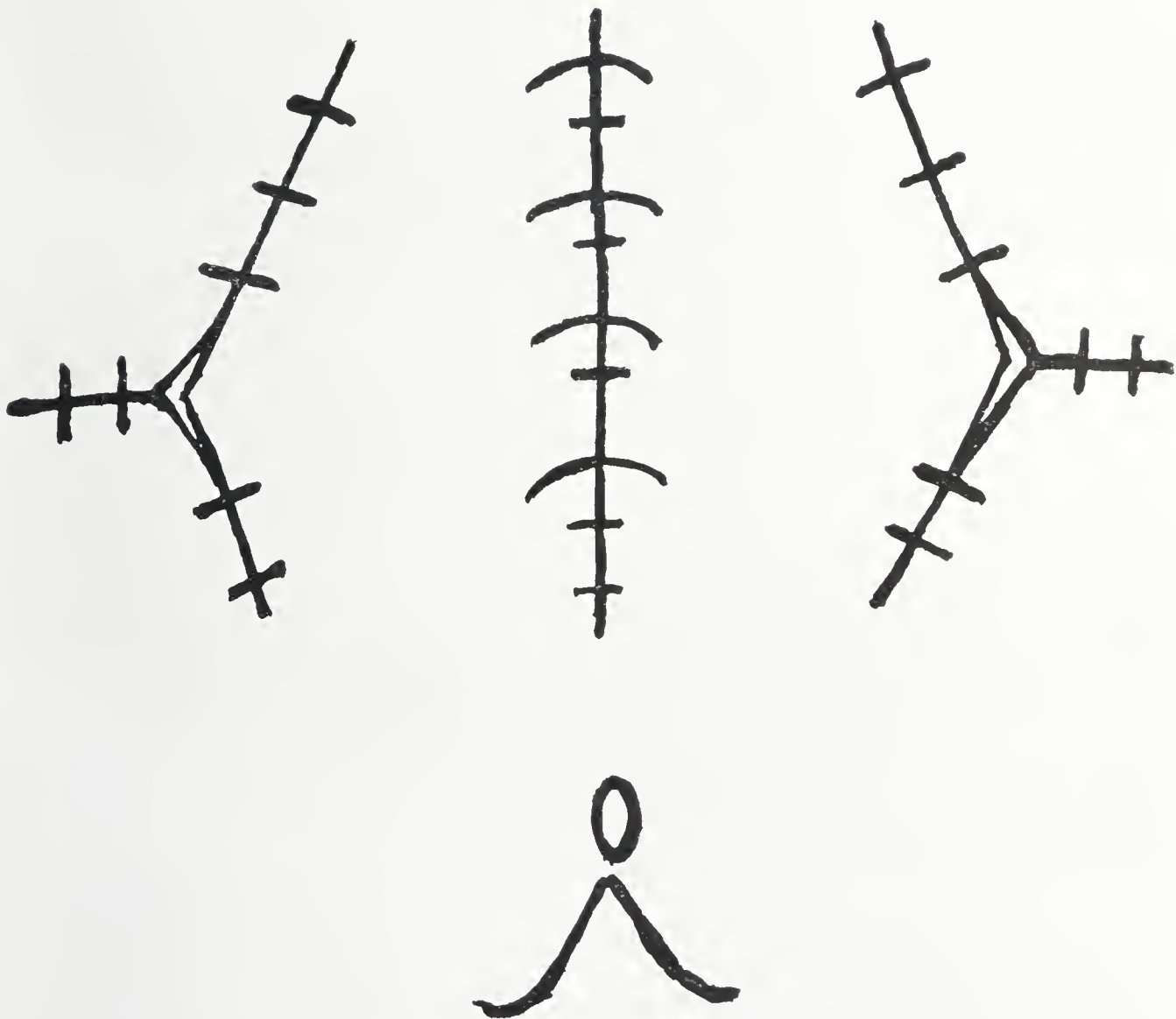


FIG. 10

COELIAC DISEASE

By D. P. BRYCE

"There is a kind of chronic indigestion which is met with in persons of all ages, yet it is especially apt to affect children between one and five years of age. Signs of the disease are yielded by the feces; being loose not formed but not watery; more bulky than the food taken would seem to account for; pale in colour as if devoid of bile; yeasty, frothy, an appearance probably due to fermentation; stinking stench often very great, the food having undergone putrefaction rather than conoction. . . . To diarrhoea alba add emaciation and cachexia and we have the complete picture."

This is Gee's classical description to which we have added little in fifty years. However, further study has given us a broader conception of this disease and it is necessary therefore to more clearly define the subject. One must also bear in mind that the picture presented in the literature is that seen in the hospitals, if more severe and advanced than that met with in practice in this country, where the cases are treated early and not allowed to reach such dire straits.

Firstly, almost identical clinical pictures are found in sprue and non-tropical sprue or ideopathic steatorrhoea by which it has come to be known in recent years. Thaysen in 1929 reviews this question presenting many conflicting opinions and evidence. He observes that there is no condition present in coeliac disease that has not also been reported in sprue and ideopathic steatorrhoea and vice versa; that although these conditions differ greatly in that

(a) Coeliac disease is a condition affecting children in the temperate zones.

(b) Sprue is a disease of adults in the tropics.

(c) Ideopathic steatorrhoea is a disease of adults in temperate zone, often preceded by no obtainable evidence of earlier gastro-intestinal symptoms.

Nevertheless such distinction is by no means absolute. Rather than to insist on identity he believes that these conditions should be grouped together as the "ideopathic steatorrhoeas". Recent work has not changed this conception.

Secondly, Miller and Perkins in 1923

differentiated a mild or non-diarrhoeic type. This characteristically shows:

1. Milder symptoms with more insidious onset.

2. Stools which though larger than normal are darker than in the acute type, not so offensive and contain only 20-50% fat, chiefly in the form of soaps.

They consider this to be true coeliac disease because the essential feature is still the failure of fat absorption and on low fat diet stool fat though improved is still definitely higher than normal. More indirect evidence is obtained by study of the quiescent stage of the acute type which presents a similar picture.

Thirdly, Anderson of New York in 1938 reported a case of his which gave a typical picture of coeliac disease, was treated favourably for such a diagnosis, but which at autopsy demonstrated cystic disease of the pancreas. Stimulated by such a finding the author reviewed, 45 cases of cystic disease of the pancreas of which 25 survived two years of life before dying of pulmonary infections and which were recognized as precoeliac or diagnosed as coeliac; and also the 32 cases of coeliac disease reported in the literature on which post-mortems were done. He concludes from such a study:

1. The clinical picture of coeliac disease can be presented either by:

(a) Cases with pancreatic insufficiency who survive the first year of life.

(b) Cases with no essential abnormality of the pancreas.

2. The two conditions can be differentiated with certainty only by examination of the duodenal contents for enzymes or by microscopic examination of the pancreas. Clinically the pancreatic types demonstrate the disease from birth, more frequently die early of respiratory infection and have a definite vitamin A deficiency, seen clinically.

3. Coeliac disease is therefore a clinical syndrome presented by more than one disease entity.

In this discussion we will consider only

those cases occurring in children without pathological change in the pancreas. Miller in 1926 suggested this definition "Coeliac disease is a digestive disorder arising in infants and young children who show no evidence of chronic intestinal or other organic abdominal disease; it is characterized by a persistent malabsorption of dietetic fat which causes large stools containing an excess of split fat, enlargement of the abdomen, lack of growth and other symptoms."

CLINICAL FEATURES

1. *Character of the feces.* Normal feces consist of food residue, bacteria, material secreted from the walls, leucocytes and desquamated epithelial cells. Fat, protein and carbohydrate of the diet is nearly all absorbed so that food residue is an unimportant element in the feces. Fecal nitrogen is largely endogenous as is the fecal fat which resembles the blood lipoids. Ca, phosph. and magnesium are derived largely from the blood. Normal fecal fat does not exceed 30 per cent of the total feces and 60 per cent of this is in form of neutral fat, 40 per cent fatty acids.

In coeliac disease there is a high percentage of fat in the feces ranging from 30-70 per cent. Of this the fatty acids form the greatest component indicating good splitting of fat in the intestine. A much larger per cent of soaps are present than normal and in the mild cases Miller believes that these predominate. Ca, phosph. and magnesium are high, both absolutely and relatively while nitrogen is sometimes high in acute stages.

The ammonia content of the feces is considerably greater and according to Brown, Courtney, Davis and MacLachlin this is indicative of abnormal putrefactive activity.

The typical feces are soft and mushy though usually formed, pale and frothy, often with an acid reaction and very large. As Miller has pointed out, due to secondary infection, they may become more coloured and contain blood and mucous later in the disease. As regards the acidity, which may be altered by diet, it is interesting to note the findings of Brown and Tisdale in 1924 on the acidity of an

infant's stool. They conclude that the reaction of the stool depends largely on:

(a) Ratio of CHO to protein in the diet in degree of fermentation.

(b) Type of bacteria present although these organisms may be merely the result of change of intestinal conditions rather than their cause.

(c) The amount of intestinal secretion with alkaline reaction.

(d) The extent of the zone of fermentation which as it rises in the small gut reduces absorption of CHO and increases fermentation.

(e) Also the intestinal cells require a certain amount of CHO present to stimulate their absorption so that low CHO diet may increase fermentation.

In the mild cases described by Miller the stools are large and well formed, soapy, coloured, homogenous, not markedly offensive and with an only slightly increased fat content. Miller also concludes that the pallor of the stool is no indication of the absence of bile pigment as its presence may be masked by a high fat content.

There is not a true diarrhoea in the typical case as the stools are not watery nor is the frequency often greater than 3-4 times daily.

2. Distension of the abdomen is a typical feature. It is large but not tight, being described frequently as soft and boggy. Such a condition is probably due to distension of the gut with feces and gas as well as a hypotonia of the gut wall and anterior abdominal wall. Miller has described large fat deposits in the abdominal wall and in the omentum.

3. Great wasting is a feature of the severe types. This is in great contrast to the abdominal distension. It is much more marked on the limbs than on the face which in some cases may be quite full. Associated with this wasting is a flabbiness and weakness of the muscles often associated with inability to stand and walk. Miller in 1927 points out the diagnostic value of gluteal wasting which he claims to appear early, be out of proportion to wasting elsewhere, and not to be present in other diseases likely to be confused with coeliac disease.

4. Failure of proper development forms a very important feature. This is a failure not only of growth in weight but also in length. Still in his lectures of 1918 presents the following cases:

(a) Actual weight 7 lbs. when it should be 27 lbs.

(b) Actual height 37 inches when it should be 43.

Untreated these cases are permanently stunted.

5. The nervous manifestations of this disease are attributed by Hecter to endogenous intoxication, which makes the child irritable, cranky and most disagreeable during a characteristic attack. Otherwise the patient is inclined to be introspective, quiet and thoughtful. He will often be thought to demonstrate superior intelligence perhaps because of his apparent youth; at any rate they do not show mental retardation beyond that due to enforced invalidism.

6. As far as I have been able to find there have been few X-ray studies of any importance on these children other than those of May and McCreary to which reference will be made later. However, in 1939 Kantor did extensive studies of cases of "ideopathic steatorrhea". Assuming them to be at least similar to coeliac disease, and such a suggestion is supported by the literature, his findings should be reviewed here. He finds:

(a) Changes in the small gut, especially the jejunum, of tone and motility. Particularly is there a disappearance of the distinctive shadows of the circular "valvulae conniventes";

(b) "Moulage sign"—smooth filling of upper jejunum where the valve shadows are missing;

(c) Dilatation of the colon;

(d) Faint filling of the G. Bladder; and

(e) Dwarfing deformity and osteoporosis of the skeleton.

7. *Deficiency Symptoms*—Vitamin A is a fat soluble vitamin found especially in fish liver oils, butter, egg yolk, spinach, carrots and others. Its deficiency in man leads to axerophthalmia and skin troubles as well as to night blindness.

May and McCreary studied in the fall

of this year the Vitamin A and carotenoid levels in the blood. It was demonstrated that not only were these fasting levels low, but after a test meal the blood level failed to rise normally. This being true it would be expected that definite deficiency symptoms would appear. However, axerophthalmia is never seen and skin lesions, though present, are not common or constant. Many authors claim a generalized lowering of resistance to infection which may or may not be significant.

Vitamin B complex. The absence of B₁ which is available mainly in yeast and wheat germ, and extracts such as marmite as well as in liver, kidney, legume cereals and tomato, leads typically to Beri Beri or peripheral neuritis, which may be mainly paralytic and atrophic in type or which may also demonstrate anasarca. The other significant element in the B complex is nicotinamide or the anti-pellagra factor. The features of this disease are dry exfoliating dermatitis symmetrically seen on the exposed areas of the skin, stomatitis achlorhydria, nervous and mental changes with eventual paralysis. This vitamin is found mainly in yeast and liver and kidney. Both the above factors are water soluble and both have to do with carbohydrate metabolism, and moreover it is evident that the deficiency picture of both is very close to that of coeliac disease.

Haas in 1929 discusses this question in order to show that coeliac disease is not infantile beri beri. He finds that severe cases of coeliac disease may have all the symptoms of beri beri—namely edema pallor, dyspnoea, cardiac dilatation, paresthesia, weakness, irritability and paralysis. However, he concludes that although avitaminosis B₁ doubtless contributes to the symptomology, nevertheless the two conditions are not identical because:

(a) There are cases of coeliac disease which do not demonstrate beri beri;

(b) Although feeding of B₁ as found in the banana and otherwise, produces marked alleviation of symptoms, the absorption defect is not improved by such therapy.

It is difficult to believe that there could be an avitaminosis B₁ without deficiency of nicotinic acid. Yet little reference is found to this in the literature. There are several skin conditions reported, some symmetrical and on exposed areas but others more of a generalized eczema which is particularly difficult to cure. Achlorhydria of mild degree and nervous changes are frequently present, but definite findings are lacking.

Vitamin C or ascorbic acid obtained mainly in fruits and vegetables has been shown to be definitely deficient. Its absence leads to scurvy, an essential weakening of the endothelium of the blood vessels.

Parsons in 1932 described scurvy as a frequent complication of this disease manifesting itself by bleeding into the skin, pain in the limbs, blood in the stools and urine, pseudo paralysis and swelling of the joints. As early as 1918 Still described a purpura independent of the degree of emaciation. It is interesting to consider vitamin K also in this respect.

Vitamin D is most strikingly deficient. Its absence is probably a contributory factor in the failure of absorption of calcium as indicated by its low blood level (5-10 mgs. per cent) and by the frequent tetany seen in these cases. Such tetany is most obvious during severe diarrhoeic periods. Probably the high fat content of the feces also contributes to the loss of calcium as soap. The phosphorous blood level is variable but is also usually lower than normal.

As might be expected rickets is a constant feature. Almost every author has reported such findings but Parsons has studied it most extensively. He finds genu valgum, enlargement of the epiphyses and the wrist and ankles, skull and chest signs, deformity of the long bones and multiple fractures, to a greater or lesser degree in severe cases. In the absence of demonstrable rickets definite osteoporosis is present in all cases. Rachitic changes occurs at two main stages:

- (a) In infancy when the child is still growing;
- (b) From 5-7 years of age when even

if untreated some growth may take place.

Such findings are not present in every case or both in the same case. Parsons believes that the osteoporotic bones are rachitic but as long as the child does not grow, then he is absorbing sufficient vitamin D and Ca to prevent gross changes. With growth, however, the added need results in more definite clinical rickets. An interesting feature constantly reported is the finding of transverse lines in the metaphyses indicating periods of temporary cessation of growth.

Rickets in coeliac disease then is of the low calcium type due to an absorption defect. Such cases are not cured by cod liver oil but by prolonged treatment with ergosterol and ultraviolet light.

8. *Anemia*. The pallor of these children has been noted from the first, but not until 1931 did Vaughan and Donald Hunter classify the anemias of Coeliac Disease. There are three types:

A. Usually a hypochromic microcytic anemia responding to iron therapy.

B. An erythroblastic type characterized by a very low Hb, a low colour index, and an enormous number of nucleated red cells.

C. A hyperchromic megalocytic type which shows an absence of definite poikilocytosis, RBC 2-3 mill, Hb 68 per cent and free HCl in the stomach. Up to that time only twenty cases had been reported. Recent work indicates many more. This type of anemia is found also in sprue, gastrocolic fistula, tropical anemia and has the following characteristic differentiating it from Addisonian anemia:

- (a) Indirect V.D. B. is low;
- (b) Poikilocytosis is not a feature;
- (c) Free HCl is present in the stomach;
- (d) Reacts favourably to liver but also to marmite, a vitamin B₁ extract which P.A. does not do.

9. There are other non-important general symptoms such as a mild pyrexia with occasional night sweats and fevers.

COURSE AND PROGNOSIS

Without treatment the child pursues a course of recurrent bouts of severe diarrhoea and prostration marked by progressive emaciation and stunting of

growth. Often such a child succumbs either to exacerbation of the disease or an intercurrent infection. If he survives, improvement takes place before puberty and he may go on to a enfeebled stunted maturity. Figures on such untreated cases are not available.

Even with treatment along the conventional lines it is a slow process, often marked by remissions which may be fatal. Neale Smallwood and Shippam in 1935 reviewed 18 typical cases. They found the maximum morbidity about 2 years of age. In these cases clinical and biochemical cure as determined by blood and stool examination usually resulted after 2-6 years of strict dietetic control, but clinical recovery may precede by 1 or 2 years the disappearance of all excess fat from the stool. With the recognized therapy to be described later, treating anemia with iron, and rickets by vitamin D and ultraviolet radiation, little dwarfing remained at puberty; normal physical outline, skin, teeth, and sexual development were obtained. Hardwick in 1939 reviewed 73 cases, of which

- 33 per cent died during a exacerbation of the disease;
- 37 per cent obtained clinical and biochemical cure;
- 10 per cent were still active;
- 5 per cent recovered clinically but still showed steatorrhoea.

These cases he referred to as "latent", believing that many of them later broke down and were diagnosed ideopathic steatorrhoea arising in adults without previous history of diarrhoea. Hardwick concluded that coeliac patients tend to remain dwarfed without intellectual impairment.

MORBID ANATOMY. Many theories of etiology have been presented, based on pathological change, these will be briefly reviewed later.

Suffice it to say that there is no known pathology primary for this disease, as we have defined it.

METABOLISM. Ever since Gee's original paper failure of fat absorption has been considered the essential feature of coeliac disease. Figures have been given by Herter and others claiming that only 80-

90 per cent of fat in the diet is absorbed rather than the 90 + per cent of the normal. Our modern conception of the source of fecal fat would question the accuracy of such studies. Obviously the possible sources of fecal fat are:

1. Unabsorbed food fat which is not a factor normally.
2. Some derangement of intermediate fat metabolism whereby great quantities are excreted into the gut.

And obviously also the only work of value would be that which compares the food fecal and blood fats as to character composition and iodine numbers. Fat is split mainly in the small intestine into fatty acids and glycerine by the action of the pancreatic lipase. The soaps formed by the alkaline secretions and the bile salts have a emulsifying action on the fat increasing its surface area and reducing indigestion and solubility. This process is not impaired in coeliac disease. The fats are well split always and numerous investigators have demonstrated normal amounts of active enzymes and bile salts in the duodenum.

Bauer writing in 1928 quotes the work of Hill and Bloor and Sperry and Bloor who showed the fecal fat to be similar to the blood fats in composition and melting points and to be independent of food fat on a moderate diet. He himself points out that in the normal fat intake is less than the output and that even on a fat free diet fecal fat is considerable. In the coeliac patient this fact is even more striking; also he demonstrates by blood fat estimation that absorption of fat is almost as readily obtained in these cases as in the normal. At the same time he confesses their lack of knowledge as to the factors influencing the level of blood fat. He concludes, however, that the etiology of coeliac disease must be found in those factors influencing the excretion of used fat and CHO.

Parsons in 1932 working on the same problem claimed that the I no. of fecal fat while not identical with food fats showed similar variations with dietary changes, also he shows that the fasting level in coeliac children is lower than the normal as is the range on absorption. In

normal children there is an initial rise, on ingestion of food, on the blood fat curve. Parsons postulates that this represents mobilization from fat stores of the body. This is absent in coeliac cases, which he claims disproves the previous theory, because the coeliac child would thus have no source for the increased secretion on a low fat diet. His findings therefore do not support the secretion theory, at the same time they do not seem to me to explain the high fat content on low fat diet, by the non-absorption theory either.

Thaysen in 1935 also showed a hypolipemia in the fasting coeliac case with a low blood fat tolerance curve.

Apparently this question cannot be answered at the present time until more work is done and our knowledge of intermediate fat metabolism is more complete.

Carbohydrate. Recently the role of CHO in coeliac disease has been considered to be of more importance than fat. Certainly from the first it was realized that it was the CHO which if given caused the greatest clinical disturbance. Very early it was noticed that the CHO tolerance curve was flat and the fasting blood sugar level was slightly lower than the normal in this condition.

Maclean and Sullivan of New York in 1929 summarized the problem as follows: The flat blood sugar curve could be due to

- (a) A low renal threshold—but there was seldom a glycosuria;
- (b) Lack of absorption; or
- (c) Endocrine disorder; perhaps of pituitary, adrenal or parathyroid.

Thaysen of Copenhagen in 1929 and 1935 presents the following evidence in favour of the endocrine theory.

1. Intravenous Dextrose resulted in a slight fall in blood sugar level.

2. R.Q. rose nearly to 1 after CHO ingestion.

3. Also on low CHO diet the R.Q. is still .817, indicating a reserve of carbohydrate.

Badenovich and Morris in 1936 studied this question extensively. They postulate that defective absorption may result from:

- (a) Defect in absorptive powers of the intestinal mucous membrane.

If, however, such a defect exists other crystalline substances should be equally affected. However, their experiments show that urea is quickly absorbed in normal amounts and appears early in the urine. Thus they conclude that in the absence of histological abnormalities in the wall, the condition of the intestinal mucosa cannot itself be held responsible.

(b) Physiochemical change in the intestinal contents.

Such changes may be due to the presence of great amounts of fat which prevent adequate contact of glucose with the gut wall or to the excessive fermentation of glucose resulting in its excessive hydrolysis with CO₂ production. They found that if fat and protein be fed to normal children along with glucose then the blood sugar curve becomes much flatter than normal. However, in non-coeliac steatorrhoeas the blood sugar curve is normal.

To support their other contention they show that when CHO is fed in the form of banana, the rise is double that after glucose. This they claim to be due to the fact that the invert sugar of the banana is less easily fermented.

As regards the influence of the endocrine glands the authors inject insulin into their cases and controls prior to making a blood sugar curve. They show the coeliac patients to be more sensitive to insulin than are the normal as they show a much lower blood sugar relative to the normal fall.

Finally working with anterior pituitary extract they show that by a course of injections preceding the glucose test, that the curves become normal. Thus they claim that the defect in CHO metabolism is due to a deficiency of contra-insular hormones. This work and that of Thaysen seemed to have definitely indicated the nature of the disturbance as it seems to me at least that their work is quite conclusive.

However, May and McCreary of Boston last year presented very conflicting evidence in a beautiful set of experiments. In these they consider the importance of gastro intestinal motility. As little sugar is absorbed in the stomach delay in its

emptying would affect greatly the blood sugar curve. They point out also that absorption from the small bowel depends ultimately upon the intimate contact of the mucosa with the contents, which is the result of the segmenting and "onrush" peristaltic action of the small bowel. They used barium with their glucose and simultaneously viewed the gut and constructed blood sugar curves. On the same person they did in rapid succession; oral glucose tolerance tests, tests where the glucose was put directly into the duodenum thus removing the influence of the stomach, and tests using "mecholyl" which increases the tone and motility of the gut. Their results may be summarized as follows:

1. Emptying of the stomach was delayed in coeliac cases so that usually there was 50 per cent of the food in the stomach 3 hours after ingestion. Also the jejunum and ileum were dilated; segmentary action was extremely weak and sporadic, as was also the peristalsis, so that the food tended to collect in clumps.

2. Maximum average rise in normals is 80-100 mg. per cent. Orally in coeliac cases the average rise was 40 mg. per cent. Intraduodenally 16 of the cases gave a average normal rise. In 6 with the use of mecholyl an even higher rise than normal was obtained. These 6 cases previously showed no improvement with intraduodenal (increased motility was seen in each case of improved blood sugar curve) feeding.

In the study of recovered cases the gut motility was shown to be normal.

So again we have conflicting evidence which is very convincing, and which argues that the defect is of absorption and is a result of conditions affecting unfavourably the normal motility of the gut.

Salt and protein metabolism are not primarily disturbed.

Theories of Etiology

1. Digestive disorders:

(a) Disorders of Bile. This was first suggested by Cheadle who gave the name "acholia" to the disease. Later Miller obtained some benefit by feeding his cases bile salts. In 1931 McCrae and Morris

postulated a alkaline reaction in the duodenum with absence of bile salts partially as a result of the deficiency of the enzyme "secretin". However, Neale has shown the contents to be acid, and McLure, Westmore, and Reynolds among others have shown by intubation that both bile and pancreatic ferments are present and active.

(b) Deficiency in pancreatic juice. This has been shown to occur in some cases by Bramwell and others, but such cases are not recognized as truly coeliac. Deficiency has never been demonstrated in the classical disease and indeed the stool fat is always excessively split. Such a finding, however, is claimed not to be pathognomonic in the presence of active pancreatic enzyme.

(c) Gastric Juice. It has been contended by many authors that there is a deficiency in gastric secretion in these cases. Ogilvie in 1935 confirms the evidence of impaired secretion of free HCl but shows that true achlorhydria does not occur, and that there is no reduction in peptic activity. However, he believes the hypochlorhydria to be a factor in the production of hypochromic anemia in these patients.

2. Chronic Enteritis. That the basis of this condition be chronic enteritis was the original conception of Gee. Moorhead, Payton Armstrong and Nabano and Still all published P.M. records reporting the finding of enteritis. However, their cases are doubtfully diagnosed and Miller later reviewing them, concludes that in all cases the diagnosis was incorrect or else the findings the result of secondary involvement, and of too acute a nature to explain the chronic condition. Thus he stated and it has been repeatedly confirmed since that there is no primary enteritis in coeliac disease. In the conditions marked by enteritis in which the coeliac syndrome appears the following points aid in differentiation:

(1) Wasting is more severe than the lack of growth.

(2) Abdominal wall is thin and the diseased organs may be palpated through it.

(3) Blood and mucous in the stools.

(4) Indigestion of fat is not out of proportion to other food.

Herter presents very interesting work suggesting that it is the presence of an abnormal bacterial flora which irritates the bowel. He finds almost exclusively gram positive slightly curved bacilli in the stools which he claims are present in the typical breast fed stool but not in that of a normal infant. These organisms he names *B. Bifidus* and *B. Infantilis* which are both anaerobic, acid producing in type. He also describes coccal forms responding closely to *Streptococcus lacticus*. The common *B. Coli Lactis Aerogenes* group are infrequent in pronounced Coeliac disease but become more common with improvement.

Brown, Courtney, Davies and MacLachlan in 1925 confirmed Herter's contention that there is a delay in these children, in the normal change from the gram positive or aciduric group of the breast fed in fat, to the gram negative or colon group of organisms. They do not, however, find the typical *B. Bifidus*, *B. Infantilis* stool but rather consider the organism found to be one type capable of great cultural and morphological change. That organism they refer to as *Bacillus A*.

They confirm Herter also who claimed that excessive putrefactive activity takes place and is responsible for some of the symptoms.

They showed that *Strept. Lacticus* used in the preparation of lactic acid and protein milk which is the main element of the diet, quickly establishes itself as the predominant organism of the feces, displacing *Bacillus A*. Such findings probably give a clue as to the mechanism by which such a diet improves the patients' condition.

3. Deficiency of the vitamin C complex. The most recent work carried on by May, Blackfern and McCreary have revived this theory. As will be seen before their work amply justifies such a view.

TREATMENT. From the first it was realized that diet was the "keystone" of treatment. Gee advised that fresh cow's milk not be given and that high starchy foods were very bad. He used 4 pints of

goat's milk a day or lacking this escalded whey. One of his cases did very well on a diet of best Dutch mussels. Cheadle restricted fat and CHO. Still in 1918 again pointed out the bad effect of fresh cow's milk. He used human breast milk, low fat dried milk, or goat's milk which contains less than 1 per cent of fat; restricted CHO and tended to give mostly protein. He reviewed the value of the different drugs and intestinal antiseptics and concluded that they were of little value. Such is the opinion to-day.

Howland in 1921 was the first author to publish definite work on a system of diet. He concluded:

"Halfway measures are quite unavailing and cause only loss of time, which is a serious matter . . .

The dietary treatment must be divided into three stages. First, that with protein milk alone—that more than any other food is likely to bring satisfactory results. The diet of the second stage consists of protein milk reinforced by almost pure protein foods. The duration of this stage is many months, it may be years. The third stage, that in which CHO is added is the really difficult one. The addition must be made very gradually with most careful observation of the digestive capacity. The treatment is time consuming but these patients will repay the effort expended upon them. They do not remain semi-invalids. Many become vigorous and strong."

Brown, Courtney and MacLachlan in 1927 summarized the essentials of diet as follows:

In a diet. CHO—must be avoided as far as possible.

Fat—must be reduced, however extreme reduction may actually reduce absorption as it has been shown that increase of fat on the diet increases its absorption to some extent although such results are spoiled by the acute reaction which follows.

Protein—some authors advise restriction of protein also because they believe it to cause excessive putrefaction. It was this that led Herter to advise the use of gelatin because lacking as it does, triptophane and tyrosin, it does not undergo

putrefaction to the same extent. Indol and skatol are not produced from gelatine and it can be used to replace the caloric value of CHO and fat in part. They advise a high protein diet because:

Firstly, protein curbs excessive fermentation by forming an unfavourable medium for it and by changing the reaction of the intestinal contents from acid to alkali. Thus if the presence of an acid loving flora is the cause of the condition the therapy is rational.

Secondly, by giving protein in the form of protein or lactic acid milk a large quantity of an organism (*Streptococcus Lacticus*) is introduced into the intestine which is opposed to the infantile type of Herter.

Thirdly, a diet of protein or lactic acid milk supplemented by curds provides a food which is the richest possible in calcium and phosphate.

They suggest diets for the mild, moderately severe and severe types. In the severe type they have diets A, B, C and D, to which the patients graduate as their tolerance improves. Diet A consists of 8 to 10 oz. of protein milk every 4 hours for five feedings. Such a diet is strictly adhered to until the stools have lost their acidity and show no evidence of fermentation. Then gradually the patient is built up to a full diet. The most difficult step is that of CHO addition which may so disturb digestion as to necessitate starting all over again. From diets A to D takes 18 to 24 months. This diet is used at present at the Sick Children's Hospital and is in general the universally used therapy. During its entire course cod liver oil in 10 minim doses fed gradually increasing to one drachm fed is given and due to the constipating effect of the high protein diet, laxatives have to be given regularly.

Some variations exist. Haas first in 1924 and since advocates the use of banana as a form of well tolerated CHO and experiments indicate that CHO as it occurs in banana is relatively well absorbed. Haas also regularly irrigates the gut to remove toxic accumulations.

He concludes in 1938 that the symptoms of coeliac disease depend on intolerance to CHO as a result of which fat digestion is impaired. Such a condition he believes can be readily cured by strict protein and banana diet and not by fat free diet, indeed fat is an important element in the diet.

Kirby of N.Y. in 1930 and 1931 was not successful with protein milk. He used no cow's milk at all but substituted a compound of calcium caseinate, starch and sugar for which he publishes good results.

Rice in 1936 classifies Coeliac disease with other nutritional disturbances such as beri beri, pellagra and scurvy, and treats his cases from the viewpoint of a deficiency disease. He concludes that he can give coeliac cases fat in the form of evaporated milk, sugar as wheat germ sugar, and the high protein mixture without relapse, and can cure them on a diet essentially high in vitamins.

In the summer of 1941 May, Blackfern and McCreary approached the culmination of their work which may write finis to the story of coeliac disease. Their criteria for diagnosis were firstly that their cases present the typical clinical and laboratory findings outlined above, and secondly that they show improvement under proper treatment. Assuming a deficiency of absorption of fat due to lack of some intrinsic or extrinsic factor, they fed their patients crude liver extract and the vitamin B complex.

They found a rapid improvement in fat and carbohydrate absorption with improvement in gut motility and corresponding amazing clinical recovery. That is, the course of the disease is favourably influenced by a substance or substances in the vitamin B complex and of crude liver extract. They suggest that such therapy improves the intermediary metabolism of fat, particularly that part taking place in the gut mucosa, and are working to isolate the active principle.

It is possible then that Coeliac disease may soon join the host of other maladies prevented by the proper exhibition of vitamin therapy.

ASCITES

By C. E. ROBERTSON, B.A., '43

Ascites is that condition in which there is an abnormal accumulation of serous fluid within the peritoneal cavity.

Etiology

Approximately 88 per cent. of all cases of ascites may be traced to cardiac weakness, renal disease, hepatic cirrhosis and neoplastic peritonitis. In men, cirrhosis is the commonest cause; in women carcinoma of the ovaries should be considered; in children, tuberculous peritonitis is almost always responsible.

Abnormal fluid in the abdominal cavity may be associated with the following conditions:

1. *Generalized edema:*

the result of circulatory failure due to coronary disease, myocardial insufficiency, mitral stenosis, emphysema, scoliosis or Ayerza's disease; acute and subacute glomerulo-nephritis; dietary deficiency; anemia; amyloidosis.

2. *Obstruction to abdominal veins:*

usually due to liver disease—yellow atrophy, cirrhosis, primary or secondary carcinoma, Banti's disease, haem-chromatosis, jaundice with obstruction at the liver hilum. There may be obstruction to the portal vein or inferior vena cava due to thrombosis, enlarged glands, tumours of adjacent organs, gall stones or aortic aneurism.

3. *Peritoneal irritation:*

from tuberculosis, Pick's disease, peritoneal carcinomatosis, hydatid cysts.

4. *Tumours:*

projecting secreting tumour cells from the ovary, implants of benign pseudo-mucinous cystadenoma, and fibroma of the ovary.

5. *Obstruction of the thoracic duct:*

By enlarged glands (of tuberculosis, leukemia, lymphoblastoma), filariasis, fracture of the tenth thoracic vertebra.

Pathogenesis

The fluid may be a transudate or an exudate. The complete mechanism for

the formation of transudates is not known, but the following factors play an important part:

1. *Portal hypertension and portal stasis*—especially where the ascites is due to congestive heart failure or Banti's disease.

2. *Reduced osmotic pressure* of the blood due to its lowered protein content, chiefly albumin. This occurs with parenchymatous liver disease, glomerulonephritis, anemia and protein dietary deficiency.

3. *A combination of 1 and 2.* This is the case in cirrhosis.

4. *Factors aggravating a concurrent edema*—such as capillary damage and chloride retention.

The transudation would seem to be chiefly through the walls of the mesenteric vessels. In spite of the above known facts, experimental ascites is difficult to produce. In dogs, ligation of the portal vein, even to one-fifth its natural size will not cause ascites but the formation of extensive collaterals and possibly obliteration of the portal vein.

Chylous ascites develops with obstruction of the thoracic duct in which case there may be transudation from the abdominal lymphatics or rupture of them and of the cysterna chyli.

An exudate is the same as the exudate of any serous cavity and is the result of irritation. The mesothelial cells become columnar, then cuboidal. Some may desquamate and degenerate and then be mistaken for cancer cells.

Why fluid should form in the case of ovarian fibroids is not known.

Signs and Symptoms

Normally the peritoneal cavity contains just that amount of fluid adequate for lubrication of the serous surfaces. To be detected clinically, close to one quart of fluid must be present. The increase in size and the tenseness of the abdomen are the first signs of collecting fluids.

When the amount of fluid is great: the patient stands on a wide base, with head and shoulders thrown back. The

belly is globular, the skin tense and glistening, possibly showing striae. The folds of the umbilicus have undergone an ironing out and it becomes prominent or even herniated. The superficial veins are often engorged, particularly about the umbilicus. There is widening of the subcostal angle and bulging of the lower ribs. Respiration is all thoracic and may be impaired. The heart may be shifted upwards. On percussion there is a central area of tympany with an area of dullness rising to the same level on each side. Traube's space is encroached upon. Shifting dullness is the most important diagnostic sign. Auscultation is usually of little benefit.

When the fluid is moderate in amount: The central area of the abdomen may appear flattened and the dullness takes a horseshoe shape. When the patient lies down, the bulging is of the flanks. Repeated demonstrations of even one finger's breadth of shifting dullness constitutes reliable evidence of free fluid. A fluid thrill may be elicited but the test is of little value if the presence of fluid is already doubtful. Chronic inflammation causes shortening of the mesentery and withdrawal of the mesentery to the right side so that Thomayer's sign of tympany on the right side and dullness on the left, is obtained. Pitfield's "diaphragm sign" is elicited by percussing the back of the thorax with one hand while the other feels for a palpable vibration of the Quadratus Lumborum of the same side. Fluid, if present, transmits the vibration to the diaphragm, causing the lower ribs and Quadratus Lumborum to move.

When the amount of fluid is very small: it may be all in the pelvis, or in the infracolic spaces if the patient is lying down. The abdominal wall gives a doughy resistance. Percussion is best done with the patient on one side or in the knee-elbow position when the dullness is about the umbilicus. A diagnosis may be made more difficult by the presence of areas of loculated fluid such as may occur with tuberculosis and carcinoma.

The X-ray picture is one of a compact homogeneous shadow on the flanks and a tendency for the small intestine to

show separation. The diaphragm is elevated with flattening of the dome, particularly on the left side.

Differential Diagnosis

The common possible causes of a distended abdomen have been conveniently tabulated as fat, flatus, faeces, foetus, and fluid. A diagnosis should not be made without rectal and vaginal examinations. The abdomen should be re-examined after catheterization, an enema or paracentesis. Peritoneoscopy may be helpful and a more widespread use of this method is advocated by many.

1. *Obesity*: The flesh can be gathered up. Fluid thrill is only significant if an assistant has held a hand down the midline. Shifting dullness must be relied upon.

2. *Tympanites*: the abdomen is uniformly tympanitic and peristaltic waves of definite pattern are often discernible and may at times be initiated by flicking the skin. With a pneumo-peritoneum the area of hepatic dullness is obliterated, and with pseudo-ascites (fluid in the distended coils) the line of dullness in the flanks is wavy.

3. *Faecal matter*: palpated as patchy, nodular masses. Rectal, proctosigmoidoscopic and roentgenologic examination are invaluable.

4. *Foetus*: look for additional signs of pregnancy.

5. *Distended bladder*: this may reach above the umbilicus. Catheterization should never be omitted. Note also any associated incontinence and resonant flanks.

6. *Cysts*: an ovarian cyst may fill the abdomen but the antero-posterior diameter is greater; in ascites it is the lateral diameter. The abdomen is persistently globular and the umbilicus shifted towards the xiphoid process. The flanks are usually resonant. Pelvic examination reveals the uterus drawn up and fixated with flattening of the pelvic viscera low in the pelvis. If a ruler is placed on the abdomen above the anterior superior spines and pressed towards the back the pulsations of the abdominal aorta are both felt and seen. There is no retention of bromsulphalein in the blood.

Solid tumour masses can be felt and demonstrate an asymmetrical dulness.

7. *Hydronephrosis*: note enlargement and tenderness of the costovertebral angle. The mass may show variations in size with the passage of urine. Cystoscopy demonstrates a diminution of excretion from that side. Retrograde or excretion pyelography should leave no doubt.

8. *Haemoperitoneum*: symptoms of acute haemorrhage and possibly Cullen's sign—a blue colouration about the umbilicus where there has been liver damage. Dull areas may be present about the spleen, lesser sac and below the right liver lobe.

9. *Hydatid cyst*: test for the characteristic delayed thrill using three fingers and the middle one to percuss.

10. *Chylous ascites*: Associated symptoms of starvation and copious pale fluid stools.

If a true ascites may be diagnosed it may then indicate the nature of the primary disease. The history may throw some light. Thus cancer may have associated abdominal pain, dyspepsia or gastro-intestinal bleeding; liver diseases show jaundice; and tuberculosis, a primary thoracic site. In malignancy and tuberculosis the abdomen tends to remain globular whatever the patient's position may be. Supraclavicular and umbilical nodes should be palpated for metastases. Ballottement will reveal the size of the liver and spleen. Both are likely enlarged in cirrhosis but the spleen rarely in cancer. Hepatic disease commonly has associated esophageal varices and haemorrhoids. The engorged superficial vessels show a flow in the normal direction in portal obstruction but with obstruction to the inferior vena cava the flow below the umbilicus is from below upwards. Most cases of primary liver disease develop a macrocytic anemia. Serological tests may indicate syphilis as a cause, but not exclude it, and the chest should be X-rayed for evidence of tuberculosis.

Character of the fluid

If a transudate the fluid is clear, straw-coloured, with a specific gravity between 1010 and 1015 and less than 3% albu-

min. When of recent origin, the chief cells present are endothelial. Fluid of long standing contains some polymorphonuclear cells and lymphocytes. In cirrhosis, portal obstruction and Banti's disease the fluid is darker and of higher specific gravity than in heart failure and kidney disease. The ascitic fluid of a syphilitic liver may give a positive Wassermann reaction.

When the fluid is an exudate it may be turbid. The specific gravity lies between 1019 and 1026 with an albumin content between 4 and 7%. The cause of the ascites cannot be presumed to be infective unless the cells are almost purely lymphocytic or polymorphonuclear. In tuberculosis the fluid is thin, lemon yellow or greenish. It may be bloody but the fibrin content is low. Typically there are great numbers of lymphocytes. Bacilli are not usually found but guinea pig inoculation gives adequate proof of their presence.

In secondary carcinoma of the peritoneum the fluid may be clear but it is often haemorrhagic and even chylous. The specific gravity is over 1020. Tumour cells, often of the Krukenberg type, may be demonstrated but should be in clumps to diagnose malignancy.

In chylous ascites the fluid is yellowish white and is composed of fine fat globules. Chyliform fluid is fluid that has become milky due to chemical changes and can be told from that of true chylous ascites only by microscopic examination.

In some instances the intra-abdominal fluid may urine, due to injury to the kidney, ureter or bladder.

Treatment

Treatment should be mainly concerned with the primary disease process. It is similar to that for a generalized edema and attempts to rid the body of excess fluid.

The diet is regulated to diminish the salt intake and fluids may be restricted to 800 or 1000 cc.'s a day. Saline purgatives are sometimes given and diuretics may be very effective. Among the latter are included the xanthene and mercurial types (salygran particularly), acidifying

drugs such as calcium chloride and ammonium chloride, and urea. Particular care should be taken when the cause of the ascites is renal insufficiency. (Thyroid extract and intravenous hypertonic glucose are sometimes effective in this case.) In heart decompensation, digitalis may work wonders. The value of rest, too, cannot be too fully appreciated.

When an operative measure seems advisable, paracentesis is the common procedure. A trocar is inserted in the mid-line between the umbilicus and symphysis pubis or in the flank at the level of the anterior axillary line. The fluid should be removed slowly to prevent shock which sometimes occurs with rapid removal.

In hepatic cirrhosis, epiploexy, the Talma-Morison operation has been used. The great omentum is made to adhere to the anterior abdominal wall with the object of increasing portal and systemic communications.

Summary

The commonest causes of ascites are cardiac and renal disease, hepatic cir-

rhosis and secondary carcinoma of the peritoneum. The fluid is usually a transudate or an exudate, and the most constant physical finding is a shifting dullness on percussion. Treatment consists mainly in alleviating the primary condition but removal of the abdominal fluid may be an integral part of this. Certain secondary effects may result from pressure of the ascitic fluid. Umbilical hernia may be a complication in tuberculous peritonitis of children. Snell records 49 hernias in 112 cases of cirrhosis. Occasionally when the ascitic fluid is rich in protein as in carcinoma, a generalized edema may follow.

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ARTIFICIAL PNEUMOTHORAX IN THE TREATMENT OF TUBERCULOSIS

By PERCY MINDEN, '43

Some very early records show that artificial pneumothorax was practised by the ancient Greeks in the days of Hippocrates. It was not until the turn of the present century, however, that this method of treatment was revived. Thus it is only in fairly recent years that it has become firmly recognized and established as a standard treatment for pulmonary tuberculosis.

There are several types of pneumothorax, such as artificial, spontaneous, and accidental. The artificial type alone will be dealt with in this review and it should be taken as the intentional introduction of air into the pleural space. It is currently employed in the treatment of various lung conditions, but we are here considering only its use in the treatment of pulmonary tuberculosis.

Tuberculosis is a chronic systemic disease with an inherent tendency to heal. On this fact is based the main principle of treatment—REST. To get the maximum benefit out of rest, it must be local as well as general, and in producing collapse of the lung, either total or selective, artificial pneumothorax has come to the fore as the method of preference for the therapeutic achievement of lung rest.

Physiology

We will first review briefly some important chest physiology, the appreciation of which must be complete before the mode of action of pneumothorax can be understood.

When air is injected into the pleural space, the equilibrium of the entire thorax is altered. The visceral pleura comes away from the parietal pleura and some of the pulmonary elastic tissue is relaxed. There will be therefore, less pull against the inner surface of the chest wall, less upward pull upon the diaphragm and less lateral pull upon the mediastinal septum on this side. So when the elastic tissue of one lung is relaxed the other lung will exert the stronger pull, and the mediastinum, pro-

viding there are no restraining adhesions, will be displaced away from the side into which air has been introduced.

Normally the negative intrapleural pressure is created by the tendency to recoil, of the elastic tissue of the lung. Any alteration in the size of the hemithorax, by altering the degree of stretching of the elastic tissue of the lung will alter the intrapleural pressure. The size of the hemithorax is normally controlled by the respiratory movements of the bony thorax, by alterations in the position of the diaphragm and by the displacement of the mediastinum to one side or the other. Consequently when as in expiration, the chest is smaller, the pulmonary elastic tissue is partially relaxed and the intrapleural pressure is therefore less strongly negative. In inspiration it is obviously more strongly negative.

When diseased portions of the lung lose volume, as is usually the case, the healthy parts of the lung must stretch to make up for the shrinkage of the diseased parts. The elastic tension of the healthy part of the lungs therefore is increased and intrapleural pressures become more highly negative.

However, it is possible for the healthy lung to be overstretched, in which case there will be a loss of elasticity and intrapleural pressures may become normal or subnormal. In emphysema, this is generalized and bilateral and there is a reduction in negativity of intrapleural pressures. This permits the diaphragm to be pulled down by the weight of the subjacent organs, thus creating further outstretching of the alveolar walls by enlarging the thoracic cavity.

Mode of Action

(a) *Changes in the Collapsed Lung*

The majority of evidence available points to the fact that pneumothorax collapse produces no changes whatever in healthy lung parenchyma. However, the higher the pressures employed, the

greater the degree of collapse, and the longer the duration of collapse, the more likely is it that bronchial obstruction will occur and with it fibrosis, chronic infection, and perhaps bronchiectasis.

Lung cavities are variously affected. Some cavities show complete closure, whereas there are many reports of their persistence, particularly if the cavity walls are thickened.

Fibrosis is especially evident about areas of tuberculous disease, tending to invade, as well as to wall off, areas of caseation.

(b) *Effect in Preventing Spread of Disease*

Direct extension of tuberculous disease is possible and does occur not uncommonly in the collapsed lung. It is less likely if fibrosis has had time to wall off active lesions.

Bronchogenic spread is also possible because the bronchi are not occluded. This is the probable method of origin of new foci when they do arise.

Lymphogenic spread is probably decreased because lymph flow is lessened by reduced respiratory movements. The same is true for hematogenous spread because there is a tendency for a reduction of blood flow in the collapsed lung.

(c) *Reasons for Beneficial Action*

(1) *Rest.* Rest generally has a curative value. It is obvious then that to be fully effective, the pneumothorax should completely and continuously compress the diseased areas. Maximum compression will yield the highest degree of rest but unfortunately will frequently cause serious complications. A gradual, prolonged and partial collapse is safer and more practical though theoretically less effective. Rest also lessens the absorption of toxic products and permits the mechanical factors (3) to act to better advantage.

(2) *Fibrosis.* An adequate and prolonged pneumothorax is conducive to fibrosis and healing of cavities and pericavitary lesions. It also hinders lymph flow and further prevents spread by thickening the bronchiole vascular walls. On the other hand, general pulmonary fibrosis which may occur in connection

with bronchial obstruction is not beneficial because it may later prevent the re-expansion of the lung.

(3) *Mechanical Factors.* Appositions of cavity walls and obstruction of lymph flow likely constitute the greatest beneficial effects of artificial pneumothorax.

Disease in the lung results in the loss of lung volume. Therefore about all diseased areas, the surrounding healthy lung tissue is under tension which tends to expand the area of disease. When this disease softens by caseation, the tension in the surrounding lung may cause the diseased areas to split asunder. This is probably the usual method of cavity formation.

The elasticity of the lung is reduced by disease. A pneumothorax which is still under negative pressure cannot therefore be expected to produce a great deal of collapse in the diseased lung. Actually such a pneumothorax collapses the healthy lung and this releases the diseased areas from the centrifugal strains to which they have been subjected. This appears to be the method by which the most important effects of pneumothorax are produced.

Indications for Pneumothorax

"Pneumothorax and collapse therapy in general are by no means an infallible cure, but may be expected to improve the mortality picture by 20 per cent. in the group receiving these forms of treatment." (Heaton, 1941). It should appear very obvious that there can be no hard and fast set of rules for selecting the cases that will respond to this method of treatment. Instead every patient must be considered carefully as a distinct and separate problem. Due regard must be paid to contraindicating factors and complications of which there are many. A final decision can only be made after careful deliberation where the most valuable asset is an extensive experience. The lessons taught by previous cases are of far greater value than any dogmatic list of indications that could be laid down.

Among the more common factors that influence the selection of cases for pneumothorax are the following:

1. Age: e.g., at puberty the instability of the body suggests the need for a greater margin of safety in treatment, which is provided by pneumothorax.

2. Race: e.g., among the North American Indians where greater susceptibility precludes a more grave prognosis, pneumothorax is suggested in the most minimal tuberculous lesions.

3. Activity: e.g., when tubercle bacilli persist in the sputum or there is parenchymal spread as confirmed by X-ray examination in spite of adequate bed rest treatment, pneumothorax tends to limit the activity.

4. Cavitation: e.g., when cavities are present and constitute a danger of bronchogenic spread, early and permanent closure is more often assured by pneumothorax collapse.

The following case history is typical of many in which this form of treatment is given.

Miss H. B., age 23, was admitted to Sanatorium in October, 1933. Her history was of a slight unproductive cough for the last one and one-half years, and a severe chest cold five weeks before admission following which cough became productive. On examination moist sounds were heard in both apices, and sputum was found to be positive for tubercle bacilli on direct smear.

X-ray examination showed a thick walled cavity measuring an inch and a quarter by an inch and a half in the mid-zone of the second and third interspaces of the right lung. Left lung showed a thick-walled ring shadow measuring a half an inch by a half an inch, near the periphery of the first interspace. Condition was diagnosed as far advanced pulmonary tuberculosis.

Right pneumothorax was started in October 1933, and patient became sputum free shortly afterwards. A satisfactory collapse of the cavity in the right lung was obtained and there was some definite clearing in the left lung as well. By April 1935, after gradually increasing amounts of exercise patient was discharged, and was to continue receiving her pneumothorax refills from her home doctor.

In July 1937 patient returned to sanatorium for re-examination. She

had been living a normal life for the last year and was in apparent good health. Physical examination revealed no moist sounds in the chest. X-ray showed a forty per cent. collapse of right lung and due to compression it was impossible to see the state of the lesion. Left lung appeared clear, a very fine scar being all that represented the previous lesion. It was decided to discontinue the pneumothorax by gradually extending the interval between refills and allowing the lung to slowly re-expand over a period of six months.

In May 1940 patient again returned to sanatorium for examination. She had been in good health, had gained weight and was living a normal life. X-ray showed a rounded area of shadowing in the right inner second interspace, that represented the healed area of disease. Her condition appeared inactive and was classified as apparently cured.

Discussion. The progress of Miss H. B. is typical of the course taken by many patients who receive pneumothorax therapy. It is a straightforward case in which indications were favourable, progress satisfactory and where complete re-expansion of the lung has taken place. The compression effect of the right pneumothorax on the left lung is rather remarkable although a unilateral collapse has frequently been noted to clear up bilateral disease.

Contraindications

Any condition which directly or indirectly leads to a deficiency in the oxygenation of the blood as seen in the anemias, cardiovascular disease, emphysema, etc.; any mechanical abnormalities which interfere with the obtaining of a free pneumothorax such as widespread pleuritic adhesions; any manifestations of the tubercular process that constitute an obvious threat of severe complications such as high fever, dyspnea, cyanosis, these and many other factors definitely exclude the use of pneumothorax as an adjunct to treatment.

However surgical interference sometimes obviates a contraindication, as evidenced by the division of adhesions by pneumolysis transforming an otherwise

inefficient and incomplete collapse into a successful pneumothorax.

The following is an interesting case showing the response of a patient to pneumothorax where the indications were doubtful from the beginning.

Mr. G. G., age 64, was admitted to sanatorium in November, 1936. In 1928 he had been in sanatorium for five months and was discharged with far advanced disease but negative sputum. Patient had been feeling well until January 1935 when he developed a severe attack of influenza. He had been coughing up about 4 oz. of sputum daily which was positive for tubercle bacilli on direct smear, and he has experienced gradually increasing fatigue and weakness. On physical examination chest was markedly emphysematous and some moist sounds were heard from the left lung. W.B.C. was 15,000.

X-ray examination showed a large cavity in the upper right lung field extending from the first rib to the second interspace. Left lung showed a great degree of infiltration in the upper half of the left lung and a large area of rarefaction with thin soft walls was seen extending from the first to the third ribs. Pulmonary emphysema and extensive bilateral fibrosis were quite marked.

On account of the extensive cavitation in both lungs and as a final attempt to improve the patient's condition, pneumothorax was commenced on the left side in December 1936. This was discontinued after twelve refills as no evidence of the collapse could be seen. In February 1937 a right pneumothorax was started and although a greater part of the right lung was collapsed the cavity itself could not be compressed. This pneumothorax was carried on until early in July when patient's feet started to swell. The failing heart was considered indication enough to discontinue the pneumothorax. There was gradual extension of the edema to the upper limbs, shortness of breath gradually increased and patient died on August 27th, 1937.

Discussion

Whether or not pneumothorax was contraindicated in the above case is a

matter of opinion. After considering the age, the emphysema, the general condition of the patient, and the large size of the cavities, treatment by pneumothorax could at best only have been expected to prolong the patient's life temporarily. Since the first signs of cardiac insufficiency appeared after five months of treatment, we may reasonably suspect that the extra burden of a collapsed lung thrown onto an already weakened heart was partly responsible for the onset of failure.

The indications for pneumothorax are seldom clear-cut and obvious. It is only after carefully balancing the possible risks and dangers that may arise, against the probable benefits that a decision can be made. As is seen in the above case such a decision is often extremely difficult and even after expert judgment has been made, disappointments in the progress and final outcome of the disease are more common than would be expected.

Complications

It must be remembered that even during the course of a favourable pneumothorax, when contraindications are few or absent, the unexpected nevertheless often does occur, producing a distressing if not tragic situation.

Noteworthy among the more important complications are the following:

1. *Pleural Effusion*

This is the commonest of the complications and occurs more frequently in far advanced and acute cases. It also nearly always follows damage to the underlying lung by the pneumothorax needle. Except for the possibility of causing dyspnea it is of little consequence, providing the fluid remains clear.

2. *Purulent Effusion (Empyema)*

Pleural effusions which become purulent inevitably lead to serious consequences. This complication commonly occurs in cases of far advanced pulmonary tuberculosis where there is a great degree of lung collapse. Furthermore a purulent effusion not infrequently becomes a mixed empyema as the result of secondary infection.

3. *Spontaneous Pneumothorax*

Serious results of the spontaneous or accidental escape of air from the collapsed lung into the pleural space are rare, and are seen only with the valvular type of pneumothorax or where there is secondary empyema.

4. *Atelectasis*

Pneumothorax produces partial collapse of the lung. If for any reason the diminished respiratory movement leads to collection of secretion in the bronchi, or if for any reason the bronchi become stenosed as in tuberculous bronchitis, the lung will become completely collapsed. This atelectatic state results in a tendency for non-tuberculous infection to lead to fibrosis which is the chief factor that later prevents lung re-expansion.

5. *Miscellaneous*

Mediastinal hernia, hemothorax, air embolus and acute dilation of the stomach are some of the rare complications that have been reported as producing serious consequences.

The following case history exemplifies how unforeseen difficulties are sometimes encountered during the course of pneumothorax treatments.

Miss N. P., age 22, was admitted to sanatorium in September 1935 with the following history. Patient was well until 21 months ago when she had a severe chill after skating on a cold night. Cough developed and continued with 3-4 oz. of sputum daily. In September 1934 there was hemoptosis of a cupful of bright red blood. During the past year there were frequent and sometimes severe pains through the right shoulder and in the last week through the left shoulder. For the last six weeks bowels had been irregular. Constipation for a day would be followed by two days of diarrhoea.

On physical examination patient appeared as a pale, poorly nourished young girl of 110 lbs. Pulse was 112, blood pressure 114/68, hemoglobin 54%, and W.B.C. 19,500. Sputum tests were positive for tubercle bacilli on direct smear. Heavy and coarse râles were heard in the upper right chest and were increased after coughing.

On X-ray examination of the chest, right lung showed a cavity of 2-3 cms. in diameter, above the level of the second rib. The outer third of the second interspace was also filled with an area of density. The left side showed an increase of linear markings throughout the lung particularly in the first interspace.

A right pneumothorax was started October 2nd, 1935, in an effort to collapse the cavity. In December a gastrointestinal barium series was positive for tuberculous enteritis. As it was evident at this time that adhesions were interfering with the pneumothorax, a right pneumolysis was performed producing some improvement in the collapse. However, the cavity failed to close completely and the sputum remained positive. A further pneumolysis was carried out in June 1936 and a satisfactory collapse was obtained. The next sputum tests in August 1936 were negative.

In July of the same year patient developed a right pleural effusion, and by October the fluid had become purulent. Later when the patient detected oil of peppermint which was placed in the filter through which air was introduced into the pleural space, it was evident that a bronchopleural fistula had developed as well. A right sided subcutaneous abscess also arose at the site of the empyema aspiration. The pleuritic fluid subsequently became secondarily infected and in January 1937, a right thoracotomy for permanent drainage was carried out. In February she began to develop severe attacks of shortness of breath accompanied by extreme pain over the heart. Patient continued to get worse and had frequent attacks of vomiting. In April drainage from chest became foul and more profuse. At the same time it was found that swallowed grape-juice appeared in the empyema drainage immediately afterwards. After this the patient was fed through a Rheufus tube and she became more and more listless and nauseated and she was discharged at her parents' request on May 6th, 1937. On May 24th, her pulse became rapid, feeble and irregular and she died the next morning.

Discussion

The indications for pneumothorax were fairly clear. It was a case of far advanced pulmonary tuberculosis with a secondary intestinal infection, and every effort was made to check the progress of the disease and close the cavity. Although bronchopleural and esophagopleural fistulae are quite rare, the development of empyema is a complication that rises not uncommonly though not always with such unfortunate results. There is obviously no way of telling what course the disease would have taken had the patient been put on bed rest alone and pneumothorax not attempted. Recovery would appear very questionable indeed.

This case well illustrates some of the hidden dangers and uncertainties that are forever lurking as menacing hazards on the road to a successful cure by pneumothorax.

Summary

(1) The physiological action of air in the pleural cavity has been briefly described.

(2) The mode of action of pneumo-

thorax in the treatment of pulmonary tuberculosis has been discussed.

(3) Some of the indications, contraindications and complications are mentioned and illustrated by case histories.

Conclusion

Although artificial pneumothorax is credited with striking and dramatic results in the arrest and cure of pulmonary tuberculosis, there are serious dangers connected with this treatment. A complete history, thorough physical and roentgenological examinations, and their study by one experienced in this type of therapy, should always precede the initiation of artificial pneumothorax.

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Feature Section

J. B. ROBERTS, Editor

THE MEDICAL STUDENT OF TODAY AND HIS RELATIONSHIP TO THE FUTURE OF MEDICAL ECONOMICS

By J. A. HANNAH, B.A., M.D.,

Managing Director, Associated Medical Services, Incorporated

Because the medical student of today is the physician of tomorrow; because in this transition from student to physician your minds are plastic and attuned to change; and because it appears inevitable that there will be another factor involved than was apparent in the transition period of students of yester-years, it seems appropriate that we should call to your attention this very important new development. When I was asked by your Editor to write an article on Associated Medical Services I felt that a discussion of this particular organization was much too limited. I therefore decided to focus attention on the whole field of medical economics.

It is the general consensus of opinion among those of us who have been graduated for some time that we are due for some radical changes in the economics of our profession. It is well that we should kindle in your minds a thought along this line. You who are still pliable and who yet have to pass from the embryonic stages to the full fledged growth of the physician can make the change not only more useful, but more pleasant, and easy for yourselves and the profession as a whole.

As you emerge from your classes as internes, and then perhaps at the present time, into the army, and perhaps at a later date into post-graduate work, thence into the field of actual practice, you will find much that does not fit in with the stereotype picture painted in your text books and, in some cases, given you by your professors. The degree of success which you, as individuals, will attain in the profession of medicine is dependent upon your ability to adjust to these apparent paradoxes. Much of

your happiness will depend upon your ability to draw, through experience, a constructive type of reasoning.

It is all too common a mistake for the student, and even the physician, to judge that degree of success by the amount of money which he is able to gather unto himself. This is an error which will lead to much unhappiness and false prestige, which each of you within your own hearts will know to be a betrayal of the high trust placed in the profession by the public. I would not suggest to you that the physician is less worthy of his hire than any other profession, or group of the public. Rather, I would definitely state that if you will be true to the Hippocratic Oath which you will, in the near future, be taking, you will find that in seeking first the advancement of your patients welfare, in the truest sense, all these other things shall be added: Honour, happiness, true and full life, and economic recompense. If you fail to fulfil your responsibilities to that oath by seeking only economic recompense, then all the others will eventually evade you.

So it is that I make bold to draw to your attention, a change which is bound to come, and which indeed we are told is at present in the process of being brought about. We are told that the Federal Government has an Act in regard to Health Insurance already written, and ready for presentation at the next session of the House of Commons. We are told that the Provincial Government is seeking the assistance of organized medicine in drafting an Act which will bring about the most satisfactory state of affairs, in so far as the future economics of our profession is concerned, and it is possible that such an Act will

be required before the next session of our Provincial Legislature.

With such imminent changes in the process of evolution, it is well that you, as students, and future members of the profession, should give considerable thought to the question, and know what is good and what is evil in the system which we have followed up to the present. You should realize that any change brought about should be brought about by evolution, rather than by revolution. In fact, you should know that experience has taught that, however sincere the elements may be in a revolutionary and sudden change in the practice of medicine, it has never been possible to make such revolutions operative. Although some forms of health insurance have been continuously in existence in the English-speaking world since 1912, they have developed by a process of evolution, despite all the bickerings and bitter quarrellings which have resulted from an attempt to bring such changes about by a process of revolution. So it is that I propose to discuss with you briefly, the various forms of what is commonly, and sometimes erroneously, called "Health Insurance", or "State Medicine".

After some ten years of study of this problem, it appears to me that there are three main systems included under the blanket term of "Health Insurance". It is essential to fix clearly in our minds what is meant by each one of these terms, in order that we may approach the problem with an open mind, and a view to bringing about the evolutionary changes which are inevitable, and, in fact, so essential to the welfare, not only of the public, but the profession as well. These three systems are as follows: (1) State Medicine. (2) Health Insurance—(a) Commercial enterprises, such as Sickness & Accident Insurance, and (b) government controlled health insurance. (3) Voluntary budgeting against the future costs of medical care. We shall endeavour to analyse each of these three, showing what, in our opinion, is the weakness and strength of each, and finally, endeavouring to draw some conclusions as to what should be the objective of the profession.

(1) State Medicine

Very frequently, this term is used for all the various forms of paying for medical services under any form of insurance, as opposed to our present system of individual payment and practice. This, in our opinion, is absolutely incorrect, and requires clear delineation.

In so far as we have been able to discern, State Medicine is that form of practice in which all those who have thing whatsoever to do with looking after the health of the public, or patients during an illness, are Civil Servants, and paid a salary by the State. There is only one country in the world of which we know at the present time where such a system pertains throughout the whole practice of medicine. That country is Russia. I believe it has come about in Germany to a greater or lesser extent since the advent of Hitler and his "co-thugs". A perusal of medical literature during the past two decades will show that neither of these countries has contributed very greatly to our scientific advancement, and initiative appears to be definitely curtailed. It must, however, be understood at the outset, that the true scientist will not be deterred in his ambition by any system, but it is quite probable that much which would otherwise be available to the public and the profession will be suppressed under such a system because of the political factors which enter into consideration under any entirely state controlled project.

It may be somewhat of a surprise to you and to members of the profession, to realize that for something more than one hundred years, one branch of the practice of the profession of medicine in Ontario has been under state control, namely, psychiatry. It is only necessary to look at the advances made in general medicine as compared with psychiatry, to realize that there is something definitely wrong with that system. It was my privilege for a period of seven years to work with the men in psychiatry in this province, and to do research with them. I must, however, frankly admit that I was not happy with what I saw, either in actual experience, or through a study of the history of psychiatry from

its inception in Ontario. Even now, I feel there is much under this system which is inimicable to the advancement of our knowledge of mental diseases.

I do not personally feel, and I think the vast majority of the profession agree with me, that we in Canada have any desire to see State Medicine actually brought into effect, where each of us becomes a Civil Servant on the payroll of the government and subject to the many vicissitudes which will inevitably handicap both the economic and scientific development of medical knowledge.

(2) Health Insurance

Health insurance can conveniently be divided into two parts: (a) Commercial enterprise, such as Sickness and Accident Insurance, with a profit motive, and (b) government controlled health insurance.

(a) *Commercial Health Insurance*

Let us admit at the outset that we have no quarrel with the present system of profit under properly constituted methods. I think, however, we must also admit that any system which takes as its premise that it is legitimate to make profit at the expense of unfortunate or suffering humanity, is neither desirable nor beneficial. Sickness and Accident Insurance, under a commercial type of enterprise has been in existence in this continent for at least twenty-five to thirty years, and it is the repeated experience of such organizations that they must, from time to time, and at ever shortening intervals, increase the premium until only a small percentage of the population can afford to pay such premium.

Let us not jump to the conclusion that the fault lies entirely with those who administer such commercial plans. In such a set-up, there is established a conflict of interests. The administrators have as their basic principle the legitimate and necessary project of paying dividends on the capital invested. The persons insured feel that they in turn have the right to some form of return on the money which they pay in, and if an illness does overtake such an individual, the natural tendency is to secure

as much as possible, either rightly or wrongly, during such a period of illness.

At the same time, the physician practising under such a plan is also only human, and he in turn feels that he should be compensated from a large corporation for a good deal of work which he has done in the immediate past for those who cannot afford to pay for it. So it comes about that the common attitude among the profession has developed that any insurance patients are a lucrative source of return. I think it will be admitted privately, if not publicly, that it is common practice among the profession to charge as much as they think they can on any patient where a third party is involved. Let us not be mistaken, we are not condoning such an attitude, and feel that it is entirely contrary to honest and good principles, as well as good practice.

Thus we see under any plan of Health Insurance run on a commercial basis, the primary object of the plan defeated by its own inadequacies, and inabilities, to meet the frailties of human nature. It therefore does not appear that commercial enterprise in the field of health insurance can be expected to prove satisfactory. Indeed, the fact that the problem has not been solved by such enterprises during the course of twenty-five to thirty years, seems to be indicative of what we have stated.

(b) **Government Controlled Health Insurance**

At first glance, it would appear that government controlled health insurance is synonymous with state medicine. A little thought, however, will show that such is not the case. The best example of government controlled health insurance of which I am aware, is that existing in Great Britain since 1912. Under this plan, the individual, the employers and the government all make contributions towards a common pot, out of which doctors are paid on a panel basis for their patients.

Such a plan has many commendable features, and indeed, if the indigent and low-wage-earning income group are to be included in any plan of health insurance, the assistance of the employer and

the government is essential. It must never, in our opinion, come about that any individual is provided with medical care without his contributing whatever he can towards the plan. It may be as little as ten cents a month, but that amount should be contributed under any circumstance.

Neither is there any reason why it should be presumed that the vast majority of employable individuals should not be paid sufficient to provide the basic necessities of life, if they are employed at all. I can see no reason why the government or the profession, or the public, should subsidize industry or employers to the extent of providing medical care for employees. This principle is recognized by the vast majority of employers today. There are, however, a certain number of employers who fail to appreciate the necessity of putting such a principle into actual practice.

Such a system of health insurance has its virtues, and it is fair to say that failing the creation on the part of the medical profession of a better system, it is to be expected that such a system will come about. The unfortunate part of the situation is, that the lethargy of the medical profession, both in the matter of internal discipline and control, as well as producing a better plan has, in every country so far, left the profession more or less "holding the bag", until such time as the profession caught up in their thinking to that of the public. Only after many heartburns and bitter struggles have they been able to attain some of their rights in these situations. The profession in the Dominion of Canada today are in imminent and immediate danger of allowing this situation to develop here.

It is fair to say, however, that if such a system is established beyond the vicissitudes of political influences, on a truly economical and sound basis, that it is indeed the only way in which the profession can expect to be remunerated from a large proportion of the general public on any sort of reasonable basis. It behooves you then as students, and the profession as a whole, to study this problem seriously, and know the pitfalls as well as the advantages, so that we

may bring about that form of health insurance which will prove most advantageous under the circumstances for not only ourselves, but the public at large.

(3) Voluntary Budgeting for the Future Costs of Medical Care

Some four years ago the profession of this province was faced with certain situations, and certain members in the profession felt that some development should be brought about whereby the profession would be in possession of knowledge in regard to the establishment of health insurance. As a result, there have developed in this province, a number of voluntary organizations which permit budgeting against the future costs of illness. There are three such plans which have been in operation now for upwards of four years. They are: Associated Medical Services Incorporated, which, at the present time, covers some twenty-seven thousand subscribers; the Hollinger Employees' Medical Services in Timmins, Ontario, which covers some nine to ten thousand individuals, including miners and their families; and a plan in Windsor, known as the Windsor Medical Services, which has some two thousand five hundred subscribers, according to our latest information.

At the time these organizations were established, organized medicine in this Province felt that it did not want to take either the moral or the financial responsibility of such undertakings. They were established as independent, incorporated bodies. From the experience of Associated Medical Services, it appears that it was fortunate that the profession acted in this way, since it has permitted the development of these organizations without a biased approach. They have shown the way, and there is much valuable information to be had from their experience over the past four years.

Having been managing-director of Associated Medical Services for that four-year period, I shall roughly outline the basic principle underlying our operations. For a stated sum, either for an individual or a family group, Associated Medical Services undertakes to pay for the cost of medical care for any illness

which has its origin after the individual becomes a subscriber. An individual subscriber pays into the fund \$2.00 a month and, in return, gets home and office services of a general practitioner; services of a specialist and a consultant; hospitalization in accordance with the need of the case at the prevailing semi-private rate, not exceeding \$3.50 per day; X-rays; such special nursing as is basically necessary, and laboratory procedures.

This plan has shown two main weaknesses. First, although 28% of subscribers to Associated Medical Services have incomes of less than one thousand dollars, it still leaves many of the low-income bracket with moderately large families who cannot afford to pay for the service. This group is obviously in need of assistance, perhaps both from the employer and the government. The second weakness lies in the fact that in any voluntary scheme where an individual may come in or stay out as he chooses must, of necessity, exclude from its responsibility conditions which existed at or had their origin prior to the date of application.

Neither of these weaknesses, however, is insurmountable. If we overcome the first, and have the assistance of the employer and the government for the employee, making it compulsory for individuals under a certain income level, the second weakness is automatically taken care of. If there is a wide enough cross-section of the community, as would be the case under a compulsory plan, it is possible to take care not only of illness arising in the future, but of illness which may have had its origin in the past.

The profession in Canada is definitely opposed to a panel system, whereby a physician is paid a flat sum for all services for each individual who selects him. Consequently, when Associated Medical Services was established, it was agreed that we would pay on a fee-for-service basis, in accordance with a schedule of fees as agreed upon by the Ontario Medical Association. That we have been able to carry on for a period of four years, and build up a reserve, is proof of the fact that such a plan can be successfully operated. In fact, that we have

co-operating with us more than 90% of the profession in those districts where we operate, who are, generally speaking, quite satisfied with our arrangements, is evidence of the fact that it can be made acceptable to the profession. The fact that the public continue to pay their subscriptions, and continue to subscribe in ever increasing numbers, is evidence that there is a demand and a necessity for such a service.

It is true that there are difficulties with both the profession and the public, but it is fair to say that perhaps 5% of both the profession and the public give rise to more than 90% of all the difficulty. This brings us to the consideration of our next point.

Internal and Individual Discipline

Every plan of health insurance requires strict honesty on the part of both the profession and the public. We all know that human nature being as it is, such exact and strict honesty cannot be forthcoming in 100% of cases. It is therefore axiomatic that whatever system of health insurance is evolved, there will devolve on the shoulders of the medical profession a severe and strict responsibility to see to it that its members adhere not only by lip service, but in actual act, to our Hippocratic Oath. We all know that there is a small percentage in every walk of life which is continually spoiling a good thing for the remainder, and the medical profession is no exception. It therefore becomes necessary for us to admit and recognize that some form of authoritative and efficient control must be exercised over both the profession and the public. Such a responsibility is indeed colossal. We can most assuredly expect, whether we like it or not, that the future is going to demand of the organized profession as a whole, that they exercise in practice that control over the ethics of the profession to which they at present give such glib lip service. If the profession fails, they can rest assured that either the government or the public will establish such control. It will be an unfortunate day for the profession, if they persist in ignoring this basic principle. It therefore behooves you, as students, to know and

understand the organizations which control discipline in our province. It behooves you, the moment you graduate, to associate yourselves with, and closely follow, all activities of both your local and provincial medical organizations, and the College of Physicians and Surgeons.

Finally, I think it is but fair to give consideration to the problem of the amount of control which the profession should expect over the economics of the practice of medicine.

It is an admitted fact that the medical profession is, or should be, in a position to know what their services are worth. Unfortunately, it frequently occurs that the profession is entirely unaware of how much the public can afford to pay for such services. For example, it is generally conceded that a childbirth, and the pre-natal care which goes with it, is a much more difficult and time-consuming procedure than a clean appendix operation. Yet it is a fact that the vast majority of children are born in the Province of Ontario at an average fee of somewhat less than \$25.00 per birth, whereas the average appendix probably yields around sixty to seventy-five dollars. It appears that there is an obligation on the part of the profession to maintain the cost of childbirth at a low level, otherwise our birth rate will be seriously affected. In my four years' experience with Associated Medical Services, I have had many a young couple join the organization and tell me that up to the present they have not been able to afford to have a family, but now that they can budget against the cost of such a procedure, they are going to go ahead and have children. It would appear that the profession has indeed a very serious responsibility in regard to such matters.

This, then, brings us back to a consideration of the latter point which we have just discussed, namely, the exercise of control over our economic desires, and internal discipline of such a nature as will make it possible for the public to secure their necessary medical attention. The ever increasing cost, as our knowledge advances, complicates this factor still further. We must, however, pre-

sume that the profession has, or will gain in the future, that knowledge and disciplinary power which will entitle them to their just degree of control in any form of health insurance which may be brought forward.

We must see to it that the profession controls the method of practice. That is, within the region of just discipline, a physician must be allowed to treat his patients in accordance with the necessities of the case irrespective of cost. The profession should not only retain control over research, but should be in a position to collect the cost of such research as a part of any plan of insurance. In our opinion, it is long since past the time when the profession should have to beg for the finances with which to do research. It appears to us that the cost of research should be a definite part of the cost of health insurance, and an appropriation set aside for that purpose.

In order to ensure such control, any commission, committee or organization set up should have a majority, but not too large a majority, of the profession in such controlling body. Such body must, of necessity, be subject to the rigid discipline of our code of ethics and our Hippocratic Oath.

Finally, I would like to say a word on the broad principle of approaching your new experiences.

Having had some experience in the field of psychiatry, one is aware of the havoc which fear can play in the normal thought processes. If you, as students, and we, as a profession, can approach any problem without fear of the unknown, but rather like the Knights of the Round Table approach it in the manner of a glorious adventure, there is nothing in either the professional side of medicine or the economic side, either as at present constituted, or as it may develop in the future, which can not be that glorious adventure. Our profession is traditionally conservative, and it is well that it should be. We are dealing in sums of life and death, states of health or illness, all of which vitally affects the future welfare of our patients. If we will approach this and every other problem unafraid of the future, knowing that the

past has shown that we are capable of meeting such situations, there is nothing which we cannot tackle with confidence.

Let us not, however, confuse the issue of conservatism and fear. In our experience, conservatism has been beneficial, in that it has been necessary for us to prove our point beyond the question of a doubt to our seniors, and those of greater experience. There is, however, a fine line between conservatism and fear. Sometimes the fearful would have us believe that they are applying the principles of conservatism when in actual fact they are shielding their fears. The

greatest weakness which we can have is fear. Nothing is ever static except what is dead, and death is so final that we cannot afford to allow Shakespeare's quotation to apply to ourselves, namely, "Cowards die many times before their death".

To sum up, then, you students who will soon become the members and directors of our profession should give earnest and serious thought to the development of the future. If you do so without fear, the problem will add another drop to the overflowing measure of a happy experience.

Alumni Notes

By T. S. WILSON, M.D.

Seemingly the fears of conjugal psychopathy have not deterred the Class of "41" from marriage, for no fewer than sixteen have crossed the great divide, and, according to all reports, are doing as well, or better, than can be expected. Aside from these sixteen, three were already married, old stagers at the game as it were, and two more we know of (God knows how many more!) are seriously contemplating it. Which all goes to show that Meds 4T1 are on the whole a home-loving lot, and care more for the solid comfort of fireplace and slippers than the casual misdeemeanors that brighten a bachelor's existence.

Not only to take up space, but because they have really done something, the names of the newly married ones are set down forthwith.

Two girls head the list—Alice Whiteside and Laurie Patten. Ben Guyatt should follow the latter closely. Then, there are the two Bell's, Clint and Bob, John Scott, Lloyd Hisey, Bill Ortved, Jack Denne, Bob Wilson, Murray Townsend, Dave Albertson, Ted Watson, Tom Black, Bob Demary and Bill Howe. The last one's demise is so recent, we may be guilty of a Winchell act. Roy Forsey is so close to getting married that he may as well be mentioned in the list. Congratulations to all of them; may the gods be kind.

Aside from marriage, only four of our class have strayed from the beaten path of interneship—Lloyd Hisey to enter the Navy, Roy Forsey to take an assistance-ship at Collingwood, Ted Watson to work for the C.I.L. at Nobel, and Murray Townsend to take a job as doctor for some mining company in far-off B.C. Lloyd is rumoured to be stationed in Newfoundland at present. It is noteworthy that these hardy souls have all become married, which just goes to show that a pioneer in a certain line of endeavour is often one in another as well.

Speaking of marriages and meaning no offence to the above, we heard a joke the other day that has a certain flavour to it. A young lad approached his girl with "Let's get married or something." She replied, "Let's get married or nothing!"

As for the rest of us, I think most are looking forward to joining some branch of the services next spring or summer. If this does not start another orgy of marriages, we have guessed wrong.

Best wishes to the Journal and its Staff for the coming year. Although we admit being biased, it is a fine institution.

Abstracts

D. P. BRYCE, Editor

Studies in Immunity to Pertussis

J.A.M.A., 1941, Vol. 117: 79

The evaluation of immunization against whooping cough is based on data accumulated through clinical observation of immunized and non-immunized children and by laboratory examinations of blood samples from vaccinated and non-vaccinated children by the opsonocytophagic test. In a group of 379 immunized children between six months and five years of age, there were 58 exposures to whooping cough with the development of 9 cases or an incidence of 15.5%. The initial series was a group of 140 unvaccinated siblings. Here there were 47 known exposures with a case incidence of 37 or 78.7%. Cases in the vaccinated group were in general milder than in the non-vaccinated group.

The blood investigations revealed that, up to 2 months of age the reaction rating was high due possibly to the placental transference of antibodies, but later the ratings were low. Children who had been vaccinated or who had previously had whooping cough had high ratings. Those who contracted the disease in the series started with a low rating which rose as the disease progressed. The authors believe that they have shown that it is of value to immunize children against whooping cough and that revaccination two years later is a desirable procedure.

R. J. W.

Ozena

M. M. Cullom
Nashville, Tenn.

J.A.M.A., Sept. 20, 1941

In this very comprehensive and instructive review, the author presents the case of ozena or atrophic rhinitis. He concludes that the foul crusting is the end result of a chronic purulent focus

which may be in the nasal cavity the accessory sinuses or the post nasal space.

It is claimed that over 90% of children suffering from an exanthem, diphtheria or epidemic influenza, show involvement of the accessory sinuses which is responsible for the purulent discharge finally ending in ozena.

Chronic rhinitis then is a disease of childhood especially in the female, and one which has lately been decreasing in incidence. This is due to proper treatment of nose and throat disorders, elimination of dietary deficiencies, and a more healthy athletic life for boys and girls.

Disposal of the purulent focus eliminates the crusts and fetor and allows the individual to return to his proper place in society.

D. P. BRYCE.

Electroencephalography

Hallowell Davies, M.D., Boston

J.A.M.A., Sept. 20, 1941

To most of us, this most recent of diagnostic means, is but a name. The author presents the subject in such a way as to make the reader fully conversant with it.

With electrocardiography, electroencephalography deals primarily with the electrical phenomena or properties of the body itself, that is, an application of electrophysiology. In contrast to heart sounds, the brain works without revealing physical signs of its activity, and thus electroencephalography which records directly the electrical output of the cortex and thus its activity, promises to be of even greater value than electrocardiography.

To be of value the electrical output of the brain must be sufficiently regular, lawful and simple, and the changes produced by disease or mechanical interfer-

ence must be great enough and characteristic enough to be susceptible of sure detection.

The first condition is partially fulfilled as the range of normal pattern can be delimited with confidence and be divided into fairly definite types. The extremes of pattern have been shown to correspond with personality differences.

The changes in pattern present with intracranial lesions, although giving no information as to their nature, will roughly localize the disturbance. This is particularly true of surface lesions. Some insight into the basic nature of epilepsy is obtained and early diagnosis of attacks may be attempted.

D. P. BRYCE.

The Freidman Test on Spinal Fluid in Cases of Hydatidiform Mole in Pregnancy

American Journal of Obstetrics and Gynecology

September, 1941

By Morton Vesell, M.D., and
Stanley Goldman, M.D.

This article by Morton Vesell, M.D., and Stanley Goldman, M.D., of the Beth Israel Hospital of New York is presented as a preliminary report upon work being done in the investigation of the use of the Freidman test upon spinal fluid in the diagnosis of hydatidiform mole.

These workers present two cases of hydatidiform mole in which this test was positive on the spinal fluid pre-operatively, and several cases of early and late normal pregnancy in which the test is positive on urine but negative on spinal fluid. They find too that ectopic pregnancy, ovarian cyst, and fibroids, in a limited series of cases do not produce a positive spinal fluid test, although one case of seminoma was found to do so.

The authors are carrying on in the hope that they can prove that a positive spinal fluid Freidman test is diagnostic of hydatidiform mole, and is not produced by normal pregnancy or other conditions.

J. G. M.

Penicillin: An Antiseptic of Microbic Origin

British Med. Journal, Aug. 30, 1941

This article deals with two antiseptics of microbial origin. The first is "gram-cedin" obtained by extraction from a gram positive organism *B. brevis* found in soil samples fed exclusively with a certain type of bacteria. This material is active in vivo and in vitro against many gram positive bacteria especially staphylococcus.

The second is "penicillin" contained in fluid cultures of *Penicillium notatum*, long used to inhibit staphylococcus in culture media. This material is also inhibitory to many gram positive bacteria, and has no noxious effect upon humans when given intravenously. It has been used with apparently favourable effect in several cases of staph. pyemia, given via intravenous route. A small inoculum seems to have the same effect as a large one, and the material is excreted in the urine, from which it can be recovered. Given per os it must be protected from acid by the use of sodium bicarbonate. Serum blood and pus do not interfere with its action, nor does it impede the action of leucocytes or fibroblasts.

Many bacteria including meningococcus *B. anthrax* and actinomycosis are susceptible in vitro to penicillin suggesting its tremendous possibilities and the need of further investigations into its effects and mode of action.

J. G. M.

Prevention—The Treatment of Choice in Pulmonary Embolism

By J. Ernest Ayre, M.D. in the
C.M.A.J., September, 1941

The time for effective treatment of pulmonary embolism is before the thrombus forms. In order to properly understand, the preventative measures to be undertaken, it is necessary that the main etiological factors be known. These are circulatory stasis and low grade sepsis. Of secondary importance are surgical trauma, secondary anemia, hyperglycaemia, dehydration, and heart disease.

Thrombophlebitis and embolism probably occur more frequently than they are diagnosed. In the puerperum or in the post operative period, very acute pain in the chest, stitch in the side, pleurisy or other chest complication, should be suspected of being embolic until proven otherwise. Over 50% of fatal embolism occur following pelvic operation.

In modern post operative treatment the patient does little for himself and the period of bed care is prolonged. These factors combined with light-breathing, abdominal distension and tight binders favour pelvic venous stagnation. It has been shown that the normal emptying time of the pelvic veins is 5-30 seconds. This is increased to 1-2 minutes in bed-ridden cases and 3½-4 minutes in the post operative cases. In the latter, stagnation is evident the day after operation and increases to the 5th or 6th day.

Low grade sepsis increases the incidence of thrombophlebitis. When a chronically infected cervix is left following a sub-total hysterectomy, it has been shown that thrombophlebitis occurs three times more frequently than when a total hysterectomy is performed.

High blood sugar and obesity are predisposing factors and should be treated with insulin and thyroid extract as indicated. Anemia, dehydration and varicose veins all require attention preoperatively.

The most important aspect of prevention is the institution post operatively of graduated exercises, definitely planned and ordered. This may be supplemented by elevation of the foot of the bed and such treatment has been shown to lower incidence from 0.3% to 0.06% in a wide series. Massage should be followed by early discharge from complete bed care.

R. J. W.

Studies On Congestive Heart Failure

The American Heart Journal, Vol. 22, No. 2

Henry A. Schraeder, M.D.,
New York, N.Y.

Up until the investigations of this writer, there had been no conclusive evidence as to whether the restriction of

salt, or fluids or both were of importance in the occurrence or prevention of the oedema in cardiac insufficiency. By the study of 23 obstinate cases of congestive heart failure an attempt was made to learn the importance of each of these substances. It was observed in all cases that a reduction in the amount of ingested salt was followed by a loss in weight and in oedema fluid.

It was also found that the intake of fluids had but little relation to the accumulation or disappearance of oedema when the salt intake was sufficiently low. Weight was gained rapidly when fluids were forced and salt not restricted but when minimal amounts of sodium chloride were given, weight was lost and oedema disappeared regardless of whether fluids were forced or restricted. In most cases a diet containing 1.0 gms. of sodium chloride were found to be low enough to prevent oedema. These findings were attributed to the fact that, water cannot be deposited in tissue spaces without salt, water per se acting as a diuretic. Salt therefore is responsible for this type of oedema. Further, restriction of fluids leads to dehydration and consequent depression in the output of urine, but as a certain volume of urine is necessary for the optimum excretion of chlorides, this condition must be prevented. So for practical reasons, the patient should be allowed to drink as much water as they please provided that the intake of sodium chloride is carefully limited.

G. G. C.

The Blood Picture in Rheumatic Fever

The American Heart Journal, Vol. 22, No. 3

Valentina P. Wasson, M.D., Edward E. Brown, M.D., and Clarice Weintrants, B.A.
New York, N.Y.

Because of the absence of a specific diagnostic test for Rheumatic Fever these workers undertook the study of the blood picture of patients suffering from the disease in an effort to obtain information which might be utilized in the diagnosis of the disease when the classical symptoms were absent. Certain nonspecific but

definite changes were found which, however, were valueless in so far as diagnosis was concerned but of value in prognosis and in estimating the general progress of patients.

Observations made on the Erythrocyte Sedimentation Rate indicated that it was the most sensitive test of Rheumatic Activity, being the first haematological abnormality to appear and the last to disappear after activity had ceased. It was so decided that patients with Acute Rheumatic Fever should not be discharged until their Sedimentation Rate was normal and that the persistence of a high value signified a grave prognosis. It must not be assumed that an increased Sedimentation Rate necessarily means Rheumatic Fever but merely infection, whose course may be followed.

The nonfilamented neutrophile count, although not as delicate a test as the Sedimentation Rate was found to be increased

in all cases but returned to normal with the clinical improvement.

In investigating the haemorrhagic phenomena frequently found in rheumatic cases, the Blood Platelets, Coagulation and Bleeding Time were studied. Over 67% of the patients were found to have a platelet count below 200,000 per cu. mm. There was an initial progressive thrombocytopenia which gave place, at the termination of the infection, to a moderate thrombocytosis. Coagulation and Bleeding Time estimations were of no consequence.

The determination of Capillary Resistance was of importance, showing a deviation from the normal in both inactive and active forms of the disease.

It appears that the haemorrhagic tendency of the Rheumatic state may be explained by (1) the Thrombocytopenia, (2) the increased capillary permeability as found in all Rheumatic cases.

G. G. C.

